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CHALMERS WATSON, M.B., M.R.C.P.E.

VOLUME I

ABDOMEN TO BONE

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Abdomen, Clinical Investigation of.

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In many respects the diagnosis of disease within the abdomen is more difficult than that of disease situated in other parts of the body. The manifestations of a lesion of the nervous system can be observed with accuracy, and its site determined largely from a knowledge of anatomy and physiology. In the case, too, of disease affecting the heart or lungs the methods of physical examination are usually adequate to enable us to determine the seat, and to some extent the nature, of the lesion. But in the investigation of abdominal diseases the means at our disposal are more limited in their application; and in making a diagnosis we have to fall back—to a greater extent than in the case of disease elsewhere—on our knowledge of pathology, and on our former experiences of cases presenting a clinical history similar to that of the case under consideration. Hence the necessity for giving careful attention to every abnormal phenomenon which is essential in the investigation of disease in any part of the body, is especially called for in the case of disease situated in the cavity of the abdomen.

The object of the present article is to take a brief survey of the main points to be attended to in the investigation of an abdominal case, and the subject will be considered under the following headings:—(A) The symptoms from which the patient suffers, together with facts relating to his previous history; (B) The physical examination of the patient; (C) The examination of the excreta, of any vomited material, and of fluids obtained by exploratory puncture.

Symptoms, etc.—In most cases of abdominal disease the symptoms from which a patient suffers are referred to the abdomen, or to disturbance of the functions of some of its viscera, but it is important to remember that this is not always the case. For example, palpitation or irregularity in the action of the heart may be the chief manifestations of dyspepsia; convulsions may be produced by a worm in the intestines; tetany is occasionally the most prominent phenomenon in gastric dilatation; and a condition similar to pernicious anæmia is sometimes associated with atrophy of the stomach. Then many abdominal diseases tend to produce mental depression, and sometimes actual melancholia. The writer can recall one case of cancer implicating the posterior wall of the stomach, in which the prominent symptoms for many months were those of neurasthenia.

On the other hand, symptoms referable to the abdomen may be initiated by disease elsewhere; thus pain in the front or side of the abdomen may be due to pleurisy or to spinal caries; vomiting may result from acute pericarditis, or from a tumour in the brain; intense paroxysmal pain in the epigastrium, associated with severe vomiting, may be the chief, possibly the only, symptoms in the early stages of locomotor ataxia.

These observations show how necessary it is to make a careful study of all the symptoms presented by a case, and how important it is to keep our minds open and free from bias, not forgetting that pain in the abdomen may depend on lesions elsewhere, and that nervous phenomena such as convulsions, vertigo, or mental disturbance may be closely related to disease of some abdominal organ.

The doctrine of *referred pain*, which has been carefully studied by Ross, Mackenzie, Head, and others, is one of great importance, and will be noticed in the various articles dealing with diseases of special organs. In addition to the site of pain, *its characters* and *associations* require investigation. As regards abdominal disease, pain reaches its greatest severity in acute peritonitis, and in the form of colic in cases of intestinal obstruction and of renal, or biliary, calculus. *The time relations of pain*, whether constant, intermittent, or periodic, its connections with the taking of food, with vomiting, with the passing of urine or fæces, are also points to be elicited. Thus the pain of uterine disease is often periodic; that of renal or biliary colic may begin and end abruptly; that of gastric ulcer usually ensues some time after taking food; while that dependent on cancer, or on aneurysm, may be more or less constant.

In a similar manner the occurrence of vomiting, any peculiarities relating to digestion, to micturition, or to defæcation, are other questions that may require detailed investigation.

Then inquiries as to the *mode of onset* and course of the symptoms, before the patient came under observation, often throw light on the nature and situation of the lesion. Thus the abrupt onset of colicky pain while the patient is walking or performing an action may be the earliest sign of acute intestinal obstruction; gradual and progressive weakness and wasting is often a striking fact in the history of abdominal cancer or tuberculosis.

Questions should also be asked regarding the *previous health of the patient*, or any former illness, or any operations that may have been performed. A history of syphilis, for example, may give a clue to the nature of a nodular liver; a history of symptoms pointing to previous attacks of intra-abdominal inflammation may elucidate the cause of an obscure case of intestinal obstruction, and so on. The habits, occupation, and place of residence, should also be ascertained. In illustration it will suffice to mention the relation of cirrhosis of the liver to over-indulgence in alcohol; the colic and symptoms of intestinal obstruction sometimes due to lead-poisoning, and the bearing of residence abroad on the possibility of an obscure abdominal swelling being an hydatid cyst, or of an enlargement of the liver being caused by an abscess.

Examination of the Patient.—Before proceeding to examine the abdomen, it is desirable to make observations with respect to the general appearance and condition of the patient—to take the temperature, to feel the pulse, to inspect the tongue, to observe *the colour and expression of the face*, the attitude of the body, and the *state of nutrition*. Details regarding these points will be given in future articles, but I would emphasise the importance of beginning the examination by a careful inspection of the patient. For example, in many affections of the abdominal organs, the face expresses irritability or profound depression; in acute peritonitis it looks pinched and withered; in severe ulceration of the bowels and in malignant disease the expression known as *risus sardonicus*, in which the upper lip is elevated, making a deep furrow on each side, curving down from just above the ala nasi towards the angle of the mouth, is often very conspicuous. Drawing up of the lower limbs suggests severe pain in the abdomen, as from peritonitis; when one leg is drawn up there may be renal or biliary colic, or appendicitis, or localised pelvic inflammation. Again, much emaciation points to malignant disease or to tuberculosis; marked prominence of the venous radicles of the face with a sallow skin suggests hepatic cirrhosis; jaundice suggests disease of the liver, or in its vicinity; general dropsy indicates Bright's disease.

Inspection of the Abdomen.—The abdomen may look perfectly healthy, even when one of its organs is extremely diseased, but as a rule a lesion within its cavity is accompanied by some change—it may be a slight one—in the colour, form, or movements of its surface. On the other hand, the abdomen may look abnormal when its contents are healthy, in consequence of some general disease, or of a lesion in another part of the body.

Whenever possible, the patient should be first examined in the standing posture, the abdominal walls being carefully examined behind as well as in front. This method is of great value, especially as regards the question of symmetry, and should always precede examination of the patient when lying down. The chief points to be observed are:—(1) *The shape and size of the abdomen.* A uniform general enlargement may be due to ascites, to meteorism, or to a great enlargement of a single organ, as of the uterus in advanced pregnancy. A local swelling may be caused by distension of the stomach or a portion of the bowel, to the presence of a new growth, to enlargement of the spleen, or to a distended bladder. A diminution in volume, a flatness or retraction of the abdomen is sometimes noticeable, as of the upper part when there is obstruction at the cardiac end of the stomach. (2) *The movements of the abdominal parietes.* In addition to the movements of respiration, the following may be visible:—Peristaltic contractions of the stomach or intestines; pulsatory movements of the aorta as seen in anæmia and in aneurysm; the movements of the fœtus in the gravid uterus. (3) *The condition of the skin and of the superficial veins.* Pigmentation of the skin occurs during pregnancy, in Addison's disease, and in other forms of chronic abdominal disease, as tubercle, cancer, and malaria. White or bluish lines (*lineæ albicantes*), together with a bluish-white colour of the general surface, are met with as a result of great or repeated distension of the abdomen. A dilatation of the superficial veins points to engorgement of the portal system, which may be caused by disease of the terminal branches of the portal vein, as in hepatic cirrhosis; by disease of, or pressure on, the trunk of this vein, or the inferior vena cava. In the last case the veins of the lower limbs become enlarged, and there is generally some œdema. The superficial veins of the lower part of the abdomen are often very conspicuous in cases of chronic dilatation of the stomach. The position and appearance of the umbilicus should also be noticed, and, lastly, the usual sites of herniæ should be carefully examined.

Palpation of the Abdomen.—In the investigation of abdominal disease, palpation is by far the most valuable method of examination. To carry it out efficiently the abdominal muscles should be as relaxed as possible, and this is best attained as follows:—The patient should lie on his back with his head and shoulders well raised and supported, and his knees drawn up. The examiner's hands, previously warmed, should be placed gently on the abdomen, and then pressed in evenly and firmly while the patient is engaged in conversation, or after he has made a forcible expiration. In examining the lateral regions of the abdomen it is useful to place one hand over the anterior wall, and the other hand over the posterior wall, of the abdomen; the latter is then pressed forwards against the hand in front, when any unusual structure or swelling lying between the two hands may be readily felt. This procedure is of special value in the examination of the spleen and kidneys. It may also be necessary to turn the patient on his side, or to examine him when he is resting on his elbows and knees. *The administration of chloroform* is occasionally required to relax excessive tension of the recti muscles, and this is perhaps especially the case in children, when sometimes deep-seated glandular mischief may be discovered, which was not perceptible before chloroform was given. Success in palpation is also much aided by previously emptying the bladder and rectum; in suspected cases of cancer the repeated use of enemata to empty the bowel is of much help in diagnosis. Sometimes, when there is obstruction in some part of the alimentary canal, it is useful to artificially distend the stomach or intestine with gas.

By palpation we ascertain the existence and site of tenderness, of resistance, of œdema, of fluctuation, and of any local swelling or tumour. The various points to be observed in connection with tumours will be mentioned in a succeeding article,[1] but I would here allude to the necessity of observing the presence and position of *the sensation of increased resistance* to palpation, for it may be the only palpable sign of a deep-seated lesion; for example, increased resistance over the upper portion of the right rectus muscle is often a prominent sign in cancer of the pylorus. A great increase in the resistance of the abdominal walls is noticed in general peritonitis; a local increase in resistance may be due to a localised peritonitis. But it must be remembered that hard tense muscles are also met with in hysterical or neurotic subjects apart from internal disease. Frequently, too, they are associated with distinct swelling, caused partly by local distension of the intestine, and partly by muscular contraction. But these "phantom tumours" come and go, and usually disappear when chloroform is administered, or even sometimes when gas is removed by means of a tube passed into the rectum.

In many cases valuable information may be obtained by *examination per rectum*, especially by the bimanual method—that is, with the finger of one hand in the rectum, the other hand being placed over the lower part of the abdomen. In this way the condition of the pelvic organs, the lower lumbar vertebræ, the glands, and any masses in the abdominal cavity below the level of the umbilicus, can be investigated, and in the youngest child as well as in the adult. The following conditions, amongst others, may be felt in different cases:—*tubercular masses or bands* in the iliac fossæ, or stretching across the abdomen; *cancerous masses;* enlargement of the *lymphatic glands;* inflammatory thickening about the *vermiform appendix*, or in connection with the lower portions of the small intestine; thickening of the *vesiculæ seminales*, which would suggest the tubercular nature of an otherwise obscure case. *Examination per vaginam*—with the finger only, and bimanually—is also of service in the investigation of disease affecting the female generative organs and neighbouring parts. In the examination of the uterus and bladder, special instruments, as the catheter and sound, are sometimes required.

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[1] "Abdominal Tumours, Diagnosis of."

Mensuration.—This is used to determine the girth of the abdomen, and thus any alterations in its size, as in cases of ascites, may be recorded from time to time. In certain cases it is desirable to measure the distance of the umbilicus from the ensiform cartilage, the symphysis pubis, or the iliac spines.

Percussion.—This method is serviceable in mapping out the size of the liver or spleen, and in obtaining the height to which the stomach note is obtained in the left hypochondrium, as well as the height to which a distended bladder rises above the pubis.

As regards the note obtained by percussion over the alimentary canal, this will obviously vary greatly at different times in relation to its contents, and to the tension of its walls. The note over the transverse colon is commonly higher in pitch than that over the stomach, but if the latter contains but little gas, while the colon is distended with it, it may be impossible to define the greater curvature which forms the line of separation between the two organs.

Auscultation of the abdominal organs is comparatively of little value. Swallowing or gurgling sounds may be heard over the stomach; friction sounds over the liver and spleen from peritonitis; a systolic murmur over the aorta in cases of abdominal aneurysm; and the fœtal heart sounds or murmurs over the gravid uterus. The method of combined percussion and auscultation is useful in defining, in doubtful cases, the lower border of the stomach, and of differentiating the neighbouring intestine.

The physical and chemical characters of vomited matters, of the urine and fæces, of fluids obtained by exploratory puncture, together with the results to be obtained by a bacteriological examination, will be considered in subsequent articles. It is sufficient in this place to emphasise the value of such examinations as aids to diagnosis.

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1. Contusion of the Abdomen

General Considerations (Anatomical and Physiological).—The importance of injuries of the abdomen consists mainly in their liability to produce simultaneous or consequent damage to the viscera or the peritoneum, resulting in death from shock, hæmorrhage, or peritonitis.

Fortunately about sixty-five per cent of abdominal injuries are not associated with any internal lesion. Of the remainder, about fifteen per cent make a good recovery after slight injury to the viscera, as suggested by evidences of peritonitis; and only about twenty per cent are complicated by distinct symptoms of serious intra-abdominal mischief.

Common Causes of Injury.—Speaking generally, the superficial injuries are associated with slighter degrees of force, such as direct blows, kicks, spent bullets, or moderate crushing; and the more serious injuries are usually produced by some severe crush, such as occurs in the passage of a heavy waggon over the abdomen, and in "buffer-accidents," or by a violent and localised blow, as from the kick of a horse.

Many cases are, however, on record where a comparatively small amount of force has resulted in rupture of one or more of the viscera; and in no case can external appearances be relied upon as a guide to the extent of internal mischief.

Nature of the Lesion.—The lesion in the abdominal wall may consist of simple bruising; considerable extravasation of blood from laceration of the parietal vessels; ruptured muscle, chiefly the rectus; or the tissues may be more or less pulpefied, and sloughing of the parts may ensue.

The lesions of the viscera consist of bruising, or partial or complete rupture. Every abdominal viscus—solid and hollow—has been ruptured, but those situated more deeply, such as the pancreas and duodenum, are less commonly the seat of injury than those more superficially placed. The omentum, mesentery, and great vessels may also be torn; in the last case the veins are more often affected than the arteries, because of their thinner walls.

Ruptures of the kidney, ureter, and urinary bladder are considered under their respective headings.

Modifying Circumstances.—There are many conditions which modify the effect of injuries on the different organs. The liver, from its friability, its large size, and its fixation by ligaments, is very liable to rupture. Such viscera, as the stomach and bladder, which are more stretched and in closer apposition with the abdominal parietes when distended, are more susceptible to injury than when in an empty condition. On the other hand, the intestines, by their free mobility, frequently escape from the effects of even severe blows. The presence of disease—such as a malarious spleen, or the pathological softening associated with ulceration or growths—renders the organs affected more liable to rupture. Laceration of the stretched ligaments, or partial or complete rupture of the uterus, may occur from injury during pregnancy. The more fixed the organ, and the greater the stretching of its ligamentous attachments (from distension, etc.), the greater its liability to injury. To this rule, however, the duodenum and pancreas, from their deep position in the abdomen, are notable exceptions.

Causes of Death.—Ruptures of the solid viscera usually cause death from shock or hæmorrhage, whereas ruptures of the gastro-intestinal tract are more often fatal from the occurrence of septic peritonitis caused by the diffusion of their contents. Many forms of bacteria are normally found in the stomach and intestines which, under favourable circumstances, multiply rapidly and assume an increased virulence. Of these organisms the Bacillus coli communis is the most important, and it is chiefly owing to its presence that the peritonitis is due in cases of rupture of the gastro-intestinal tract. The escape of the contents of the hollow viscera (gall-bladder, urinary bladder) may also result in peritonitis, though their contents are usually sterile; but the inflammatory condition in these cases is a secondary result of the lowered vitality of the peritoneum, which allows the entrance of bacteria from the intestines into the escaped fluids where they then multiply.

Means of Protection.—The manner of protection from injury of the various viscera has already been indirectly alluded to when speaking of their mobility or fixation, position in the abdomen, etc. The solid viscera are further protected by the bony walls of the thorax and by the spinal column, but a considerable portion of the intestinal tract is almost entirely dependent for its protection upon the resistance of the soft tissues only. This resistance varies to some extent with the thickness of the parietes; but the most important protection is afforded by the contractions of the anterior abdominal muscles, which, in the case of a well-developed person, are capable of offering a very powerful resistance to a blow for which there is time to prepare. The position of the body (such as flexion) by which the blow may fall on the bony walls of the abdomen, and the preparedness of the recipient of the injury when the anterior abdominal wall is struck, are amongst the chief means by which the abdominal contents are protected.

Influence of Nervous System.—The connection of the numerous sympathetic plexuses in the abdomen with the cerebro-spinal nervous system is one of supreme importance in the production of the phenomena of shock in association with abdominal injuries; and the fact that the shock in these cases bears no necessary relationship to the severity of the lesion demands the most cautious treatment in all contusions and wounds of this region. Blows upon the epigastrium are specially serious; and death has taken place from such accidents, though no intra-abdominal lesion was discoverable. The speedily fatal issue under these circumstances doubtless results from profound irritation of the nerves of the subjacent solar plexus inducing a most intense form of shock; but the mental effects of terror may be regarded as an important contributory factor in many instances. On the other hand, shock may be absent or markedly delayed, in cases where an injury, incompatible with life, has occurred—of this there are numerous examples.

SYMPTOMS AND DIAGNOSIS (*General*).—After the receipt of an abdominal injury the patient usually complains of considerable pain, and exhibits the phenomena of shock to a greater or less degree. There is an anxious expression of countenance and restlessness, the surface of the body is cold and clammy, the abdominal walls are rigid, the pulse is frequent and feeble, and the respirations are superficial.

Great thirst is frequently complained of, and vomiting is a common and early symptom. The persistence of vomiting is indicative of visceral complications; and should it contain blood it is suggestive of a lesion of the upper part of the gastro-intestinal tract.

Pain is frequently excruciating; and, though it may radiate in various directions, is often more intense over the seat of mischief. It is, however, an uncertain symptom—for if the shock is profound there may be little pain; and in most cases, after the lapse of a few hours it tends to diminish. Local tenderness, which is a constant symptom, is therefore more reliable as a guide to the injured area. Some degree of swelling is generally present in the abdominal wall, and in cases of great extravasation of blood between the muscles this may be considerable. The temperature is usually below normal, but cannot be relied upon as an indication of the patient's condition.

Although valuable information may be obtained from a history of the accident, the examination of the clothing, or the position and nature of external signs of violence, accurate diagnosis of the existence or absence of intra-abdominal mischief is frequently impossible at this stage. The variability of shock has been mentioned. In most cases of injury affecting the abdominal parietes only, it is usually slight, but it may be profound or may even be immediately fatal. The essential point to be remembered is that in contusions without visceral injuries, shock, even if profound, is usually of a transient nature, and signs of improvement are most often manifest in the pulse and general condition in the course of a few hours. In the case of contusions associated with intra-abdominal lesions, it is usual for shock to be well marked, but there are numerous instances in which it has been sufficiently delayed to allow the patient to walk a considerable distance with such conditions as ruptured liver or intestines; and it may be said that these exceptional cases of slight shock with severe visceral mischief are more common than the exceptional cases of profound shock with no such complication. Even when delayed, shock comes on sooner or later if intra-abdominal lesions are present; but whether it appears immediately, or after an interval, it always tends to persist, or to relapse after some slight improvement. It is this continuance of the phenomena of shock which helps to mark the contusions which are associated with visceral lesions.

Should the conditions of shock be approaching a fatal issue the pulse becomes irregular or almost imperceptible, and the surface of the body livid in colour. If death does not occur from shock within a few hours after the receipt of an injury which has produced an intra-abdominal lesion, symptoms of intra-peritoneal hæmorrhage, or of peritonitis, are usually manifested.

In hæmorrhage—which commonly occurs after rupture of the solid viscera or of the great vessels—the phenomena of shock become increasingly greater from the time of the accident; or, if any improvement is shown in the condition of the patient, it is soon followed by the occurrence of further hæmorrhage, resulting in the production of what Bryant has aptly described as "relapsing collapse." This gradual accumulation of blood in the peritoneal cavity is marked by great pallor and coldness of the surface, flickering pulse, sighing respiration and a feeling of breathlessness. The abdomen becomes soft and more or less distended, and the blood tends to accumulate in the flanks, where it causes an increasing dulness varying in position with the attitude of the patient. The pupils become widely dilated, unconsciousness supervenes, and death occurs in a condition of extreme collapse.

In other cases the patient may survive the effects of shock and hæmorrhage, and may die of peritonitis. This is more common after rupture of the hollow viscera, though it may occur apart from any such lesion. The symptoms of peritonitis will be considered later.

Such, then, are the general symptoms occurring in contusions of the abdomen with and without visceral complications; *but they vary widely in different cases.* When intra-abdominal mischief exists, the presence of certain special symptoms forms the most reliable guide to the localisation of the lesion. These symptoms will now be considered.

SYMPTOMS AND DIAGNOSIS (*Special*)—*Lesions of the Stomach.*—There is severe burning or gnawing pain over the site of the stomach. Partial ruptures, involving the peritoneal surface, are accompanied by vomiting, and followed by symptoms of localised peritonitis. Lacerations, involving only the mucous surface of the organ, generally cause profuse hæmatemesis. In complete ruptures, the escape of gas from the stomach into the general peritoneal cavity may cause the normal liver dulness to disappear. This phenomenon is by no means always present, and is of value only in the earlier stages; for, later on, it is apt to be confused with the tympanitic distension of the intestines, caused by the peritonitis resulting from the escape of the stomach-contents into the abdominal cavity. The drum-like distension of the abdomen produced by the passage of hydrogen gas by means of a stomach-tube, as mentioned under wounds of the abdomen, may serve to establish the diagnosis of complete rupture; but in the absence of this lesion the stomach itself becomes distended, and there is no free escape of gas into the general cavity. In most cases of complete rupture, death occurs within two days from shock or septic peritonitis.

Lesions of the Intestine.—There is good reason for supposing that partial lacerations may occur, but we have no certain means of diagnosing them. In complete ruptures, shock is generally present to a marked degree, *but there are many exceptions.* Persistent local pain, thirst, and vomiting—with or without abdominal distension—are amongst the most characteristic of the symptoms. The vomit, at first, consists of the contents of the stomach; which, later, are mixed with bile. If the patient lives long enough —generally some time during the second day—the vomit may become fæcal; but this is usually a concomitant of the peritonitis which has developed as a result of the extravasation of the intestinal contents: such extravasation occurs in seventy per cent of the cases.

Constipation is generally present, and tympanitic distension from the escape of gas into the peritoneal cavity may lead to a diminution of the liver dulness, as in the case of rupture of the stomach. The presence of free gas in the peritoneal cavity in cases of gastro-intestinal ruptures is also said to produce a greater distinctness in the heart-sounds and respiratory murmur over the abdomen. The application of Senn's hydrogen gas test (p. 15) *per rectum* may be used for diagnostic purposes. Blood may appear in the stools in rupture of any part of the gastro-intestinal tract; but, as it generally occurs late, it is not of much value in diagnosis. In ruptures of the duodenum and colon extravasation of fæces may occur behind the peritoneum and lead to the formation of an abscess.

These symptoms usually occur in association with ruptures at the time of the accident; but, in some cases, they are delayed for some days, and are then due to a perforative ulceration as a result of a previous contusion. Most cases of ruptured intestine die within two days; the mortality, without operation, being 96 per cent.

Lesions of the Omentum and Mesentery.—Injuries of these parts are usually accompanied by severe collapse from intra-peritoneal hæmorrhage —the symptoms of which have been already considered. Of fifteen cases of laceration of the mesentery collected by Curtis, all but three were fatal within twenty-four hours. Should death not occur from hæmorrhage, thrombosis of the mesenteric vessels may lead to gangrene of the bowel.

Lesions of the Liver.—A rupture of the liver may be inferred if there is great pain in the right hypochondrium radiating to the scapular region, superficial respiration, increase in the area of liver dulness, and signs of a gradually increasing intra-abdominal hæmorrhage—more marked locally at first on the right side. Extreme pallor and collapse are usually present. Jaundice may appear if the patient lives more than twenty-four hours, and glycosuria has been said to occur. If death does not result from shock or hæmorrhage, an abscess may form in the neighbourhood of the rent, or a more or less general peritonitis may ensue. The late development of many of the symptoms renders the diagnosis somewhat difficult. Though very serious, rupture of the liver is by no means always fatal—the mortality, without operation, being about 85 per cent.

Lesions of the Gall-Bladder or Biliary Ducts.—These injuries are rarely capable of separate diagnosis from the rupture of the liver, which they usually complicate. There may be jaundice, dark-coloured urine, and clay-coloured stools, if the bile is poured out into the abdominal cavity through a torn duct. The escape of bile within the peritoneum may, in exceptional cases, be recognised by the signs of free or encysted fluid in the abdomen. Edler gives the general mortality of these cases as 74·2 per cent—death commonly occurring from peritonitis.

Lesions of the Spleen.—In rupture of this organ there is great pain in the left hypochondrium, and increase in the area of splenic dulness. The symptoms of severe internal hæmorrhage are usually well marked. In extensive laceration death quickly occurs from hæmorrhage into the general peritoneal cavity; but, in other cases, the blood may be extravasated into the distensile capsule of the spleen, and the fatal issue may be considerably delayed or averted. In the latter case an abscess may subsequently develop. Rupture of the spleen usually occurs with other abdominal injuries, but in uncomplicated cases the mortality is said to be about 65 per cent.

Lesions of the Pancreas.—It is not possible to mention any symptoms specially referable to rupture of this viscus, as it has always occurred in connection with severe injury to other organs, and death has speedily resulted.

Lesions of the Great Vessels.—Complete rupture of the larger vessels will be associated with rapid and severe collapse from extravasation of blood. Should death not occur from hæmorrhage, or should the laceration not involve all the coats, gangrene may set in; or an aneurysm may subsequently develop.

Lesions of the Kidney, Ureter, or Urinary Bladder.—These will be considered under their respective headings. All that can be said here is that the presence of hæmaturia may suggest the possibility of rupture of one or other of these organs.

PROGNOSIS.—Abdominal injuries associated with rupture of the viscera are, as we have seen, fatal in the great majority of cases; but, by timely operation, if the condition of collapse is not so severe as to render it unjustifiable, many of them can be saved from death; thus, of 62 abdominal sections for rupture of viscera collected by Sieur there were:—38 cases of rupture of the intestine, with 20 recoveries; 17 cases of rupture of the liver, with 7 recoveries; and 7 cases of rupture of the spleen, with 1 recovery. The mortality in these cases of rupture of the spleen seems unduly high, for Ledderhose records 28 cases of partial or complete splenectomy for injury to the spleen, all of which recovered.

The prognosis in cases of ruptured viscera is not so favourable when operation is performed after peritonitis has commenced; and it may be stated in general terms that *the longer the operation is delayed the greater will be the mortality*; for death usually occurs within forty-eight hours after the receipt of the injury in cases of this nature in which no operation is performed.

The causes of death are shock and hæmorrhage in the earlier, and peritonitis in the later stages.

Contusions unassociated with visceral complications end favourably, unless death should occur from the immediate shock; but the subsequent formation of a ventral hernia as a remote consequence of sloughing of the parietes or rupture of the rectus muscle, must be borne in mind.

Treatment (*General*).—It will be gathered from what has been said as to the impossibility of relying upon the degree of shock as an index of the severity of the mischief, that it is desirable to treat any abdominal injury, though supposed to be of a simple nature, with the most careful attention. The treatment of shock should be the first consideration. The patient must be placed with the head low, surrounded by hot bottles and blankets. Strychnine (gr. $\frac{1}{20}$) alone, or in combination with digitalin (gr. $\frac{1}{30}$) or ether (ʒj)—when administered hypodermically—is one of the best means of combating shock. Two ounces of brandy mixed with warm water may be administered by the rectum; though it is necessary to exercise a wise discretion in the use of any stimulant, if the condition is due to hæmorrhage. All fluids by the mouth must be withheld, lest there be some visceral complication, such as ruptured stomach, which would allow the escape of fluids into the peritoneal cavity. In urgent cases hypodermoclysis —half a pint or more of normal saline solution at a temperature of 100° F. being injected into the submammary tissue—or transfusion may be resorted to.

A hypodermic injection of morphine will relieve pain and improve the pulse, but it should be avoided in severe degrees of shock. Fomentations may be applied to the abdomen if pain is great, provided there are no signs of profuse hæmorrhage.

The bladder may require attention, as retention of urine is not uncommon during the stage of shock. Moreover, the use of the catheter is desirable in most cases where the shock is profound, for by this means we may arrive at a diagnosis of the cause of the symptoms.

The accumulation of blood between the muscular planes from laceration of the deep epigastric artery is difficult of recognition; but if the signs were sufficiently definite it would be justifiable to make an incision with a view to tying the vessel above and below the seat of injury.

If, after the adoption of this mode of treatment, the patient gradually improves, and it becomes evident that the effect of the contusion is limited to the abdominal wall, the shoulders should be raised and the thighs flexed so as to relax the muscles, and the case treated on ordinary principles.

On the other hand, where the symptoms are indicative of severe intra-abdominal hæmorrhage or rupture of a viscus, cœliotomy should at once be performed, unless the shock is so profound as to produce cyanosis, an extremely low temperature, and an imperceptible pulse—death being clearly imminent.

With obvious indications of visceral mischief it is easy to know what to do; but the unreliability of the significance of mere shock, which has repeatedly been insisted on, leaves us frequently in doubt as to the best course to pursue. In such cases we should carefully watch the progress of the symptoms for a time; and, should the patient get steadily worse, or should the phenomena of shock show no signs of improvement in the course of twelve hours at the most, it would be wiser to institute an exploratory cœliotomy without further delay. Little harm is likely to accrue from such an operation—even should it be found that it was unnecessary,—whereas a further prolongation of the time of operative interference, in cases in which a severe intra-abdominal lesion exists, is fraught with danger. If delay has, unfortunately, occurred until peritonitis has developed, operation is by no means hopeless, though such cases are far less favourable than those undertaken soon after the accident.

The incision in the parietes should be made as nearly as possible over the viscus affected; but where the localisation of the lesion is uncertain, a median incision is the best for exploratory purposes.

TREATMENT (*Special*).—The abdomen being opened, and the lesion found, the necessary reparative measures will vary according to the seat of injury.

If the STOMACH is found to be ruptured, the perforation should be closed by a double row of Lembert sutures, after any bleeding vessel has been secured. Should the wound be on the posterior surface, it will be necessary to divide the gastro-colic omentum before closure can be effected. If there be any difficulty in finding the rent, the inflation of the organ by hydrogen gas passed through an œsophageal tube is useful. After the repair of the wound, the abdomen should be cleansed of the escaped stomach-contents by sponging, or by flushing with normal saline solution. Drainage will be necessary if the stomach-contents have been widely extravasated.

If the INTESTINE is believed to be ruptured, but the tear cannot be found, the inflation of the bowels by hydrogen gas through the rectum, as recommended by Senn, may lead to its recognition. When found, the perforation, if small, should be closed by a double row of Lembert sutures, as in the case of the stomach. If the laceration be large, or associated with considerable contusion of the edges, enterectomy should be performed, and the divided ends united by one of the many forms of suture, or by means of Robson's bone bobbin, Murphy's button or other mechanical appliance, according to the preference of the surgeon. Should it appear that the condition of the patient is too bad for so long an operation as excision and anastomosis, an artificial anus must be formed—the opening in the ruptured bowel being brought to the surface. The cleansing of the peritoneum, and drainage—if necessary—must be carried out as in the case of rupture of the stomach.

Rents of the OMENTUM or MESENTERY may be sutured after the bleeding vessels have been secured. If a tear in the mesentery is found close to the intestine, so as to be likely to interfere with the nutrition of the neighbouring bowel, the portion of gut had better be excised, and the ends united, in order to avoid gangrene of the intestine. Severe contusions of the omentum are best treated by excision of the affected area. The abdomen should be cleared of all blood-clot by sponging. No drainage is necessary.

If a rupture of the LIVER is found, moderate bleeding from a slight fissure may be checked by the temporary application of a sponge wrung out in hot sterilised water; the rent may then be closed by means of sutures passed through the capsule and part of the liver substance on either side. If the bleeding is more severe and the laceration larger, deep sutures of catgut should be passed through the liver substance, so as to approximate the edges of the wound and exercise the necessary pressure upon the bleeding vessels, at the same time. Attempts to secure the vessels individually are usually unsuccessful, as the liver substance is too friable to allow a ligature to hold. If deep suturing should fail, or be inapplicable, the rent should be plugged with aseptic gauze, one end of which should be brought out of the abdominal wound. The gauze should be removed at the end of forty-eight hours. After the removal of all blood-clot, the abdomen should be closed without drainage if possible. Small wounds of the GALL-BLADDER may be closed by a double row of sutures; or the margins of the rent may be sewn to the abdominal incision, as in cholecystotomy. Lacerations of the CYSTIC DUCT, and large wounds of the gall-bladder, are best treated by ligature of the cystic duct on the distal side of the tear, and removal of the gall-bladder (cholecystectomy). Rupture of the HEPATIC or COMMON DUCT often permits only of gauze tamponnage, but where possible the common duct should be sutured. An incurable biliary fistula is likely to result in these cases. All bile should be removed from the abdomen: drainage is best secured by placing a tube in the peritoneal pouch below the right kidney, and bringing the outer end to the surface in the flank. Such drainage would be desirable in cases treated by tamponnage, but may not be necessary in other cases. Rupture of the SPLEEN is treated, if moderate in size, by turning out the blood-clot, and passing deep sutures, or plugging with aseptic gauze, as in wounds of the liver. If the injury is more extensive, and accompanied by copious hæmorrhage, the organ should be rapidly turned out, the vessels at the hilum seized in a clamp, and splenectomy performed.

Rupture of the ABDOMINAL BLOOD-VESSELS presents nothing special in treatment by cœliotomy. The bleeding vessels must be found, and ligatured above and below the seat of injury. No drainage is necessary if all blood-clot is thoroughly removed by sponging.

For the treatment of rupture of the KIDNEY, URETER or URINARY BLADDER, reference must be made to the articles devoted to these organs.

AFTER-TREATMENT.—Any special indication having thus been met, and the abdominal wound closed, it is desirable in most cases to allow only liquid diet for the first few days, and to supplement this by stimulating, nutrient rectal injections. Vomiting may be checked by tablespoonful doses of hot water, and thirst relieved by the injection of half a pint of warm water into the rectum. The hypodermic administration of morphine for the relief of pain may be necessary.

The diet should be gradually improved; but the patient ought not to leave his bed for the first two or three weeks, at which time the stitches uniting the abdominal incision may be removed.

Cases of suspected partial rupture of the viscera, in which an expectant plan of treatment is adopted, would also be conducted in this manner.

Subsequent abscess formation should be treated on ordinary principles.

2. WOUNDS OF THE ABDOMEN

These injuries are met with as incised, punctured, contused, lacerated, or gunshot wounds as in other parts of the body, and may be produced by various forms of blunt or sharp instruments—the nature of which may exercise considerable influence on the extent of the mischief inflicted. The time-honoured division into penetrating and non-penetrating wounds, according to whether the peritoneum is wounded or not, is all-important; for the former are more likely to involve the abdominal contents, and end in death from shock, hæmorrhage, or peritonitis.

(*a*) NON-PENETRATING WOUNDS (*General Remarks*).—These injuries do not present any very special features, beyond the fact that blood or pus may burrow extensively among the muscular planes. The phenomena of shock are as variable in these cases as they are in contusions of the abdomen. The essential point is to be certain that the wound does not involve the peritoneum; and, if necessary, the wound must be enlarged in order that all doubt may be removed. To ascertain this point the tissues must be carefully divided in following up the track of any instrument; and in so doing, we may be aided by the charred condition of the parts in gunshot wounds. All such examinations should be conducted with strict antiseptic precautions, lest any septic material should inadvertently be carried into the peritoneal cavity. The finger is the safest probe.

Treatment.—Wounds of the parietes outside the peritoneum are treated upon ordinary principles; but it is desirable that every effort should be made to secure union by first intention, and so lessen the tendency to ventral hernia from the subsequent stretching of the cicatrix. All divided muscles or aponeuroses, in the larger wounds, should therefore be accurately drawn together with catgut, and drainage-tubes avoided if possible. Where the wound cannot be satisfactorily cleansed of dirt, or septic material, it may be partially left open, in order that all inflammatory exudation may freely escape. The muscles should be kept at rest, as far as possible, by the application of strapping over the dressings.

If the wound is a large one, rest in bed is necessary for about three weeks, and silkworm-gut stitches may be left till the end of this period, unless they are setting up inflammation. An abdominal belt should be worn for some months after the wound has healed. The occurrence of suppuration must be met by early and free incision, on account of the tendency of the pus to burrow between the muscular planes (*vide* "Abdominal Abscess"). When due to the presence of a foreign body, such as a bullet, this should be removed.

Hæmorrhage in this region must not be trifled with, or considerable extravasation may occur. It is most important in all cases where there is free bleeding to open up the wound, as far as may be necessary, in order to secure the vessel in the ordinary way.

Non-penetrating wounds, complicated by rupture of the viscera, must be treated in the manner already considered for similar lesions complicating contusions.

(*b*) PENETRATING WOUNDS.—These injuries are generally caused by stab, puncture, or gunshot wounds.

General Considerations.—Stab wounds are less often complicated by visceral injuries than gunshot wounds; the rapidly moving bullet goes through every viscus, whether full or empty, whereas the slow-moving instrument associated with a stab wound may push an empty viscus aside.

Bullets passing through the lower abdomen, from side to side, are more serious than those travelling in an antero-posterior direction; it has been calculated that, in these cases, four to fourteen perforations of the intestine may occur.

Stab wounds are generally larger than bullet wounds, and consequently we find prolapse of the omentum and viscera is more common in the former. Any of the abdominal viscera may be injured; but, for obvious reasons, the omentum and small intestine are most often wounded.

Death commonly results from hæmorrhage in cases of penetration of the solid viscera or large vessels, and from septic peritonitis in wounds of the gastro-intestinal tract. Septic peritonitis is produced by the escape of the intestinal contents; but this extravasation by no means always occurs. It may be prevented by the mutual pressure of the coils of intestine, until the formation of adhesions shuts off the general peritoneal cavity from further chance of contamination; or, in wounds under a third of an inch in length, the perforation may become temporarily plugged by everted mucous membrane until preventive adhesions have formed.

The edges of a gunshot wound are, however, so liable to undergo sloughing that a secondary perforation usually occurs, even though the wound has been closed for a time by plastic lymph.

Symptoms.—Shock and pain are as variable in penetrating wounds involving the viscera as they have been seen to be in contusions of the abdomen with intra-abdominal complications. Generally speaking, however, penetrating wounds of the viscera—as in subcutaneous rupture of these organs—are accompanied by a more or less severe shock which tends to persist.

Vomiting is common, and the ejected material may contain blood—a valuable piece of evidence if the wound is in the region of the stomach. Blood in the stools, or in the urine, may also afford some indication of the character of the penetrating wound. The abolition of the normal liver dulness, as a sign of intestinal perforation, is as unreliable as we have seen it to be in contusions complicated by visceral injuries.

So, too, emphysema of the parietes in the neighbourhood of the wound is rare, and not conclusive evidence of penetration of viscera; for it may occur in any oblique wound, by the movements of respiration, apart from any gastro-intestinal perforation.

It is clear, then, that most of the symptoms which may accompany penetrating wounds are uncertain indications of the amount of internal mischief. The only positive signs of penetration are the actual escape of the viscera or their contents through the abdominal wound, and the evidence afforded by a profuse intra-abdominal hæmorrhage.

The passage through the external wound of the fluid contents of any of the hollow viscera, such as bile, fæces, urine, or partially digested food, is not common, and cannot easily occur unless the perforation in the viscus is of moderate size, and in a line with the wound in the abdominal wall.

The rapidly increasing pallor and collapse, and the other phenomena associated with severe intra-peritoneal hæmorrhage, have already been considered and need not be repeated here.

Injury to nerves, or spinal cord, will show itself in paralysis of the parts supplied.

(In order to save repetition, reference must be made to that part of the subject dealing with contusions complicated by visceral injuries, for a detailed account of the general and local symptoms just referred to.)

Diagnosis.—The positive evidence of penetration afforded by the escape of the viscera or their contents through the parietal wound, and by the occurrence of symptoms indicative of severe internal hæmorrhage, has already been alluded to.

It is necessary to be careful not to mistake a portion of sub-peritoneal fat for a piece of omentum; and it must be remembered that in wounds of the lumbar region, fæces may escape from the colon without involvement of the peritoneum. Some information may be derived from an examination of the patient's clothing and the external wound; thus, if a line joining the apertures of exit and entrance of a bullet wound passes through the peritoneal cavity, it is probable that the viscera which lie in its course have been perforated. The length of any blood-covered portion of an instrument producing a stab wound may also give some idea of the depth of penetration and likelihood of involvement of the viscera.

But all such deductions are conjectural; and, in these uncertain cases, Senn has recommended the method of inflation of hydrogen gas through the rectum or œsophagus—according to whether the supposed lesion is intestinal or gastric—for the purpose of diagnosing visceral perforation. Hydrogen gas is preferred because of its lightness, by which it always rises to the surface, its harmless nature, and its easy ignition. It is prepared, in the usual way, by means of sulphuric acid and zinc, and is collected in a rubber balloon capable of holding about four gallons. A nozzle (for insertion into the rectum or stomach) is attached to the balloon by means of rubber tubing. Pressure upon the bag forces the gas along the intestinal tract, until it meets with a perforation, when it escapes into the general peritoneal cavity, and gives rise to tympanitic resonance over the site of the normal liver dulness. This was mentioned when considering contusions with visceral complications. Should there be a parietal wound, as in the circumstances now before us, the gas escapes audibly if the parietal opening is made patulous, and may be ignited. The flame is easily extinguished by placing a wet sponge over it. In this way, then, perforation of the gastro-intestinal tract may be diagnosed before opening the abdomen; and, in the hands of Senn, it appears to have been successful. But the inflation test may fail; its failure is not a definite contra-indication to operation; and it may add to the danger through increase of shock, and forcing of fæcal fluids through the intestinal wound into the general cavity. For these reasons it is inferior to exploratory incision as an aid to diagnosis, though it is a most valuable means of help in the search for perforations after the abdomen has been opened.

Exploratory incision, then, is the quickest, safest, and most certain way of deciding whether the wound has penetrated the peritoneal cavity, and whether the abdominal contents have been injured. To this end the wound may be enlarged, under strict antiseptic precautions, in all cases of doubt; unless some obvious contra-indication to any form of operation exists.

Prognosis.—Penetrating wounds, whether stab or gunshot wounds, are extremely fatal injuries, and have a general mortality of about 80 or 90 per cent. Gunshot wounds of the gall-bladder, spleen, and intestine—not submitted to operation—are amongst the most fatal injuries; whereas gunshot wounds of the liver are not infrequently followed by recovery.

The effect of operation in reducing the high mortality of these injuries is satisfactory, though doubtless it will be further improved by more timely interference. Of 234 cases of cœliotomy for perforating gunshot wounds, collected by Morton, the mortality was 58·9 per cent; and Coley has collected 165 operations with a general mortality of 67·2 per cent. Of these last, 81 were for wounds of the small intestine, with 25 recoveries; 24 of the stomach, with 6 recoveries; 36 of the colon, with 12 recoveries; and 19 of the liver, with 8 recoveries. Amongst the 81 cases of wounds of the small intestine, there was an average of 5·4 perforations for each case. In only 9 of the 165 cases did the surgeon fail to find all the lesions present.

The influence of time has been shown by Adler, whose statistics of 154 abdominal sections for gunshot wounds show a mortality of 52·7 per cent for operations within five hours of the injury, and a mortality of 78·2 per cent in cases of operation after twenty-four hours.

Treatment.—The general treatment of penetrating wounds is carried out in the same manner as for contusions of the abdomen.

If there is any doubt about the involvement of the peritoneum, the matter must at once be decided by dissection.

With a penetrating wound, and signs of internal hæmorrhage or perforation of viscera, there can be no question about the propriety of performing cœliotomy without delay, unless the condition of the patient is quite hopeless. The duty of the surgeon to interfere is not considered, by some, to be imperative in cases in which a penetrating wound exists without evidence of visceral lesion or severe hæmorrhage.

There is, however, a growing consensus of opinion that operation should be undertaken in all cases of penetrating wound, in order that a definite conclusion may be formed as to the existence of visceral complications or not.

Considering the increased mortality caused by delay in cases of perforation of the viscera, and the fact that a very small proportion of penetrating wounds are free from such complications, the treatment of all penetrating wounds by cœliotomy seems the most reasonable—and the sooner after the receipt of the injury the better.

All operations must be carried out with due antiseptic precautions.

If any of the viscera are protruding, but otherwise uninjured, they should be returned into the abdomen after enlarging the wound, if necessary.

Should intestine which is protruding be wounded, it should be sutured or excised, according to the amount of injury, before being returned. In severe cases, where the patient is not likely to be able to bear so long an operation as enterectomy, an artificial anus must be formed. In prolapsed and lacerated omentum the injured area may be tied off, and removed, before the omentum is replaced in the abdominal cavity.

In prolapsed and lacerated spleen, the organ should be partially or completely resected, as such operations are more successful than reposition.

Where it is desirable to explore the abdominal cavity for reasons already given, the incision should be so placed as to provide the greatest possible freedom in any necessary manipulations. For this purpose it may be sufficient, in many instances, to enlarge the wound in the parietes; but for the majority of cases an independent median incision is the best for a general exploration of the whole cavity.

The exact line of incision cannot be indicated in general terms. In every case it may be inferred that the injured organs lie in the line of the wound in the parietes, and the incision should be made accordingly.

In most cases the line of incision will be in the mid-line. This is certainly best in wounds of the stomach, transverse colon, and small intestine, and in cases of severe internal hæmorrhage. In gunshot wounds, where the course of the ball has been across the median line; where the ball, entering near the middle line, passes directly backwards; or where the course of the bullet is unknown, the median incision is also best.

On the other hand, there are cases where an incision, not median, is indicated, as in wounds of the cæcum, or of the ascending or descending colon.

After incision of the parietes, the source of any hæmorrhage should at once be sought. In this we may be assisted by packing the abdomen with sponges; but, in the most severe cases, it has been recommended that an assistant should introduce his hand through the parietal opening, and digitally compress the aorta below the diaphragm, until the source of hæmorrhage is found. Bleeding from the larger blood-vessels of the abdomen, or from the edges of a wound in the stomach or intestines, may be controlled by ligatures of silk or catgut. Hæmorrhage from the solid viscera must be met by deep suturing, gauze tamponnage, or removal of the organ as mentioned under contusions with visceral complications.

After the hæmorrhage has been stopped, search must be made for perforations and any injury repaired. If there has been so much extravasation of visceral contents that irrigation of the abdomen is considered desirable, time may be saved by carrying this out while the search for perforations is being conducted. The use of hot normal saline solution in this way will also be found of service in combating shock. A systematic examination of the whole intestinal tract and all the fixed viscera cannot always be done with impunity, and we must be guided by a rational consideration of each case in deciding to what extent such examination shall be carried.

Only those fixed organs which lie in the line of injury need be examined. Thus, if a bullet has passed through the lumbar region, perforating the ascending colon on its anterior and posterior aspects, there is no necessity for examining the stomach or the transverse and descending colon.

In looking for gastro-intestinal perforations much valuable time can be saved by the use of Senn's hydrogen inflation test, already referred to; and beyond the fact that it may make it a little more difficult to return the distended intestines, there can be no objection to its use after the abdomen has been opened. In applying the inflation test by the rectum the intestine will be distended up to the point of perforation; beyond it the intestine is collapsed. In this way the lowest perforation can be found. It is then recommended that the inflation should be used (with a clean nozzle) through the perforation before closure, in order to find a second perforation higher up. In so doing pressure must, of course, be made below the first perforation, to prevent the gas travelling towards the rectum. In this way it is possible to examine the whole intestinal tract. The stomach may be similarly examined if the test is applied through the œsophagus instead of through the rectum.

Perforations of the stomach and intestines must be repaired after the manner already considered in injuries of these viscera, without parietal wound. But in gunshot wounds, large and multiple perforations are the rule, rather than the exception, and excision of the injured gut is commonly required.

Two feet, or more, of intestine may be excised if it contains several perforations which would take a long time to suture separately, and it is quicker and safer to remove a considerable portion of intestine than to perform a double enterectomy.

The greater need of enterectomy in wounds of the mesenteric border of the gut, because of the liability to gangrene, has already been referred to. Even on the convex border of the gut, wounds above two inches in length would probably demand enterectomy.

In simple suture of intestinal wounds it is desirable to bring the edges together transversely to the long axis of the bowel, in order to prevent any tendency to stricture of the gut at the seat of injury.

(For the further consideration of the repair of visceral injuries, reference may be made to the subject of contusions associated with injury to the viscera.)

The whole of the visceral wounds having been satisfactorily dealt with, any final cleansing of the peritoneal cavity is carried out, and the wound is closed, with or without drainage, according to the amount and character of the damage done. In most cases of gunshot wound it will be wise to drain; in stab wounds it is frequently unnecessary.

Nothing has been said of the removal of a bullet or other foreign body. When embedded in the parietes or the viscera, foreign bodies do not necessarily give rise to any mischief; but their removal is desirable, if it can be accomplished without adding to the risk of operation. In the localisation of metallic substances the Röntgen rays are very valuable.

After-treatment.—A hypodermic injection of morphine may be given for the relief of pain, and to lessen peristalsis.

If the stomach or upper part of the small intestine has been wounded, the patient should be fed by nutrient rectal injections for the first two or three days—only hot water, or brandy and water, being allowed by the mouth. After this period some of the various peptonised foods should be the first articles of diet by the mouth. Solid food may be given towards the end of a week. In cases of wound of the lower part of the intestine, it is not necessary to be so rigid in the exclusion of food by the mouth, unless there is much vomiting.

Flatulent distension is common, and for this a turpentine enema affords great relief. Should symptoms of incipient peritonitis appear, saline purges should be given as early as possible, in order to aid peritoneal absorption. The treatment of the drainage-tube and the abdominal wound does not require special mention.

3. Traumatic Peritonitis

This is a clinical division of the whole subject of Peritonitis.

Etiology.—Traumatic peritonitis is essentially an acute infective process of bacteriological origin. It is true that a plastic exudation occurs, after any breach of surface of the peritoneum, which frequently appears to be independent of any discoverable organism. To such an inflammation—if we may be allowed the expression—some observers apply the term "aseptic peritonitis." It is mainly reparative in action, gives rise to no symptoms, and need not be considered here; we must leave it to the pathologists as to whether "repair" or "inflammation" is the better term to use under such circumstances. The organisms most usually found in association with traumatic peritonitis are the Bacillus coli communis, the Streptococcus pyogenes, and the Staphylococcus aureus or albus. The Bacillus coli communis is normally found in the intestine, and causes most of the cases of peritonitis in which the infective process comes from within. It is by no means necessary for the intestine to be wounded in order that the organism may escape, for it can find its way through the unbroken wall of the gut, under circumstances which have resulted in a diminution of the vitality of the intestine. This may account for the occurrence of peritonitis from contusion of the abdomen without any recognisable injury to any of the viscera, and for cases following the extravasation of such fluids as bile and blood into the peritoneal cavity—fluids which in themselves are germ-free, but afford excellent cultivation media for the development of organisms which may be adventitiously introduced.

The Streptococcus pyogenes, and the Staphylococcus aureus or albus, are commonly associated with those forms of peritonitis in which the infective process comes from without, whether it be by a stab or gunshot wound, or by the surgeon's finger or instruments during operation. Of these organisms the Streptococcus pyogenes is the most virulent.

Classification.—It would, doubtless, be more scientific to make a bacteriological classification, but as we are dealing with a clinical variety of peritonitis it will be more convenient to adopt a form of nomenclature founded on the clinical and pathological aspects of the disease.

The subject will therefore be considered as

(*a*) Localised Traumatic Peritonitis.
(*b*) Diffuse Traumatic Peritonitis.

(*a*) *Localised Traumatic Peritonitis.*—This variety occurs after such accidents as severe contusions of the abdomen, though there may be no definite symptoms of visceral injury; slight ruptures of the liver, spleen, or kidney, or partial, or even complete, wound or rupture of the gastro-intestinal tract, in which no extravasation of the visceral contents occurs.

The inflammation is limited by adhesions, so that the general peritoneal cavity is unaffected. Suppuration may or may not occur; usually it does. An abscess so formed may find its way to the surface, may burst into one of the hollow viscera, or by breaking through the limiting adhesions may give rise to the diffuse form of peritonitis.

Symptoms.—The general symptoms—vomiting, pain, tympanites, constipation, and fever—resemble those which we shall presently consider in more detail in the diffuse form of the disease, but usually exist to a less marked degree. Pain is often localised over the affected area, where a sense of resistance is felt and tenderness is also present. Rigors may occur with the onset of suppuration. The formation of an abscess is associated with a well-defined indurated swelling, which gradually enlarges. Should the abscess approach the surface the overlying skin becomes red and œdematous, and finally gives way. If it burst into the intestine a fæcal fistula may form; and should it become diffuse there will be a sudden increase in the pain, and the phenomena of diffuse peritonitis may occur.

Treatment.—It is difficult to lay down rules for all cases, as our course of action must be largely guided by the probable causation.

In the early stages of most cases a few leeches, followed by hot fomentations, may be applied to the abdomen over the affected area. The bowels should be opened by enemata, and pain may be relieved by opium. It is generally best to avoid purgatives by the mouth, lest there be any unrecognised visceral lesions. In cases of peritonitis developing during the expectant treatment of severe abdominal contusions it is desirable to at once perform an exploratory cœliotomy in order that any existing visceral injury may be repaired in the manner already described. With definite evidences of suppuration the abscess cavity should be opened without delay, lest a diffuse peritonitis should occur. After evacuation of the pus efficient drainage should be employed.

(*b*) *Diffuse Traumatic Peritonitis.*—This variety is much more serious than the localised form.

It occurs after such injuries as penetrating wounds, or rupture of the stomach or intestines, whereby their contents become discharged into the general peritoneal cavity; decomposition in extravasated bile, blood, or urine after rupture of viscera or blood-vessels; or surgical interference.

Symptoms.—Profound nerve shock is the first and most alarming symptom in all cases of severe peritonitis, and the expression becomes one of intense anxiety. The pulse-rate soon reaches 120 or more, and in the early stages is small and incompressible. The abdomen is rigid and board-like—partly from reflex spasm, and partly from voluntary efforts on the part of the patient to check all abdominal movements. Respiration is thoracic in type, because of the increased pain produced by diaphragmatic movements.

Vomiting is the most constant symptom in peritonitis. Though a small quantity of fluid only is ejected at a time, it goes on incessantly every few minutes, until the patient is worn out by its persistence. At first, and frequently throughout the case, the vomited fluids are clear, or bile-stained; towards the end they are occasionally fæcal. Tympanites, or meteorism, is nearly always present, and usually to a marked degree. It is largely produced by circulatory changes in the intestine, giving rise to the production of gas in the affected bowel. The abdominal distension is sometimes very great, and the tension of the abdominal muscles is considerable. Both are liable to considerable variation, and frequently in an inverse ratio to each other.

Pain is often very severe, causing the patient to cry out; but in some of the worst cases this symptom is not present. When due to perforation, or rupture of viscera, the pain may at first be referred to the site of the organ affected; but later on it becomes general, though perhaps worst in the neighbourhood of the umbilicus. Constipation, from intestinal paralysis, is usually present. Diarrhœa may occur, and such cases may be regarded somewhat more favourably because meteorism is diminished or prevented.

The temperature is a most variable symptom. It may rise suddenly to 102° or 104° F., the ascent being often preceded by a rigor; but some of the worst cases may have a normal, or subnormal, temperature.

Great thirst is complained of, and the secretion of urine is diminished.

Towards the end the symptoms are mainly those of acute septic infection. The patient lies on his back with his thighs flexed; the face is pinched and intensely anxious, the eyes are sunken, the skin is livid and damp, and the pulse is extremely rapid or imperceptible. The symptoms are subject to the greatest variation. They usually commence within the first thirty-six hours after injury, and death occurs in about a week or ten days. Very acute cases may die in the course of twenty-four or forty-eight hours from the time of injury. It is difficult to diagnose some of these cases from mere shock or internal hæmorrhage, but in most of them the temperature is raised.

Treatment.—The use of antiseptics is, of course, most valuable in preventing a post-operative diffuse peritonitis. After operations on the abdomen, the condition known as "pseudo-ileus" may occur, in which the earliest commencement of peritonitis is shown by abdominal distension and perhaps vomiting associated with intestinal paralysis. In this stage, the administration of saline cathartics every hour until the bowels act, may avert the threatened attack of peritonitis by clearing the intestines of noxious material and aiding peritoneal absorption. Turpentine enemata are also very valuable at this early period.

When once the disease is fully established, purgatives can only be productive of harm, and such cases should be treated by cœliotomy as early as possible, after the diagnosis is made. In diffuse peritonitis following penetrating wounds, or rupture of the viscera with extravasation of their contents, and occurring either after operation for these injuries, or during the expectant treatment of doubtful cases—cœliotomy should at once be performed, unless the condition of the patient forbids it. So, too, in diffuse peritonitis after evidences of intra-peritoneal hæmorrhage cœliotomy is indicated. By operation alone can we hope to remove accumulations of blood, pus, or other material in which the virulent germs are developing.

After the abdomen has been opened, the coils of intestine should be separated, and any blood, pus, fæces, etc., removed by sponging or by irrigation. Any visceral lesion that is discoverable should be treated, if possible, in the manner already described. The various spaces which have been emptied of blood, pus, or other foreign material should then be lightly plugged with pieces of antiseptic gauze, the ends of which are brought out at one angle of the abdominal wound. The rest of the parietal wound—which is usually best placed in the mid-line—may then be closed in the ordinary way.

Such measures may suffice for cases operated upon in the early stage, though the mortality in all cases of diffuse peritonitis is very high. Even at a later stage, where the whole peritoneal cavity is severely inflamed, the disease is not absolutely hopeless, and Finney has proposed a more radical and remarkably successful method of treatment in these distressing cases—a method founded upon the belief that the great absorptive power of the peritoneum is much impaired by the presence of the large amount of plastic exudate which covers the peritoneum in diffuse peritonitis. An incision, long enough to admit of easy access to all parts of the peritoneal cavity, is made over the part from which the peritonitis is supposed to spring. The intestines are turned out into warm towels. The abdominal cavity is then mopped thoroughly clean by pledgets of gauze wrung out in hot normal saline solution, especial care being given to the pelvis. The intestines are then similarly wiped over systematically from end to end, so as to free them from all flakes of lymph. This procedure may require considerable force, and is aided by a stream of normal saline solution. The bowels are then returned into the abdominal cavity, the most affected portions being returned last, so that it may be better drained by gauze which is packed round it. The abdominal wound is then closed except at the point of emergence of the gauze.

In the rapidly fatal cases, which apparently die from acute septic poisoning with few post-mortem evidences of the usual appearances of peritonitis, no treatment is very likely to be effective. After cœliotomy the treatment of cases of diffuse peritonitis is conducted in the same manner as after operation for penetrating wounds of the abdomen.

Burns and Scalds

Burns and scalds of the abdomen are described under article "Burns."

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Abdominal Abscess.—An abdominal abscess may occur *in the abdominal parietes, in the peritoneal cavity, in the various viscera* or *behind the peritoneum.*

This article will be limited to a consideration of Abscess in the abdominal parietes, and Retro-peritoneal abscess.

Abscess in the Abdominal Parietes

General Considerations.—Suppuration in this situation may result from blows or wounds of the abdomen, rupture of muscle, or extravasation of blood; after acute specific fevers, and in debilitated conditions generally.

In other cases it is secondary to abscess in the abdominal viscera, peritoneum, thorax, or adjacent bones.

Most of the abscesses connected with bone disease are of a tubercular nature; but primary tubercular abscess of the parietes, especially between the muscles, may also occur.

Parietal abscesses may be situated superficial to, between, or beneath the muscles.

In consequence of the loose connective tissue between the muscles, there is always a tendency for abscesses in this region to burrow extensively, though they are ultimately limited by the various muscular attachments. One or more planes may be involved as the abscess finds its way towards the skin or peritoneal aspects, on either of which it may ultimately discharge.

The weakening of the abdominal walls after the occurrence of extensive suppuration is favourable to the formation of a ventral hernia. When the abscess is associated with a perforation of the intestinal tract, a fæcal fistula commonly results.

Symptoms and Diagnosis.—The usual constitutional and local phenomena of suppuration are present. When the abscess is superficial, the red brawny condition of the skin with softened centre makes the diagnosis easy; but when placed more deeply, fluctuation is not generally obtainable because of the resistance of the overlying tissues. In the latter case, the localised hardness may simulate growths in the viscera beneath; and the fact that vomiting is a fairly common symptom in these abscesses, makes the resemblance the more marked. The contents of the deeper abscesses are frequently fœtid.

Treatment.—In an early stage, before suppuration has occurred, hot fomentations locally, and the administration of salines internally, may succeed in producing absorption of the inflammatory products. In consequence, however, of the tendency of these abscesses to burrow, an incision should be made as soon as the presence of pus is suspected; and for the same reason it is necessary to adopt thoroughly efficient drainage, especially if several fistulous openings have formed. The possibility of such an abscess bursting into the peritoneal cavity also makes early operation imperative. The usual antiseptic precautions should be adopted. After the dressing has been applied, the abdominal muscles should be kept as quiet as possible by means of strapping, and the patient should be kept in bed till healing occurs. Some sort of support, as a preventive of hernia, should be afterwards worn for some months if the suppuration has been at all extensive. If the abscess is secondary to the presence of pus in some distant focus from which it has spread, it is obvious that the primary condition must be treated before healing can be obtained in the abdominal abscess.

Retro-Peritoneal Abscess

When we consider the extensive area covered by the cellular tissue behind the peritoneum, it becomes apparent that the causes of suppuration in this region are very numerous. Amongst these may be mentioned perforating ulcer of the duodenum; suppurative inflammation of the kidney, liver, spleen, etc.; disease of the vermiform appendix, cæcum, colon, female generative organs, etc.; Pott's disease of the spine or primary tubercular deposit in the retro-peritoneal tissue; empyæma; injury, etc.

Most of these will be considered in connection with the organs or diseases with which they are associated, and to these reference must be made. The looseness of the cellular tissue is favourable to wide extension of the pus, though its position is determined largely by the seat of its origin, by gravitation, and by the guidance of the lines of least resistance. Thus an abscess originating in appendicitis would be expected near the cæcum, but an empyæma which had found its way through the diaphragm might gravitate in the lumbar region.

For anatomical reasons large extravasations of pus at the lower part of the abdomen would not be likely to pass beyond Poupart's ligament, but they might travel along the spermatic cord in the inguinal canal. On the other hand, retro-peritoneal suppuration may rise to any height under the diaphragm, when it would be called a sub-phrenic abscess (*vide* Diaphragm).

Symptoms and Diagnosis.—The varied causation renders it impossible to mention symptoms occurring in all cases, beyond those common to deep suppuration anywhere, such as high fever, tenderness, sweating, and perhaps wasting, rigors, and pyæmia.

To state the matter as broadly as possible, the symptoms resemble disease of the abdominal viscera in the neighbourhood of the abscess, and from which it may have originated. This, however, makes the diagnosis difficult, and many mistakes have occurred.

The less acute forms are very insidious, and large abscesses may form, and the patient become much debilitated, before the local condition is discoverable. Œdema of the skin may sometimes point to the presence of deep suppuration; but in many cases the skin (except for any bulging that may be present) remains quite unaffected, unless the abscess is fortunately finding its way to the surface. In most acute cases a hard but ill-defined tumour forms sooner or later, and can be felt either through the anterior abdominal parietes or bulging into the loin. Should the suppuration extend under the diaphragm the symptoms of sub-diaphragmatic abscess will be found. The proximity of the intestines produces decomposition of the contents of most of these abscesses.

Treatment.—In all cases the abscess should be freely opened and the contents evacuated, with the usual antiseptic precautions. Beyond this general statement everything depends upon the origin of the abscess. Thus a perinephric abscess is best opened in the loin behind the peritoneum; but in many cases where the posterior part of the peritoneum is pushed forward, it is desirable to perform cœliotomy and stitch the edges of the incision in the abscess sac, after examination of its contents, to the parietal wound. Drainage is desirable in most cases, but is usually unnecessary in those of tubercular origin. Each case must be treated according to its anatomical position, causation, etc. The patient's strength will require support with generous diet, stimulants, and tonics.

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Abdominal Aneurysm.

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Aneurysm of the abdominal aorta is of much rarer occurrence than aneurysm of the thoracic aorta (according to Crisp, out of 551 cases of aortic aneurysm, 59 affected the abdominal aorta); the various types, the true, false, dissecting, and arterio-venous aneurysm occur, the false aneurysm being the most common.

Its *ETIOLOGY* is the same as that of other aneurysms; it occurs much more frequently in men than women (8 : 1), and affects most frequently persons between the ages of thirty and forty; a traumatic cause can often be traced, the trauma having occurred months or even years before the symptoms are pronounced. Indirect traumatic causes may also be mentioned, as in one recorded case where a bristle 5 cm. long was found in the wall of the artery, which must have passed from the œsophagus, and caused a tear in the median coat of the aorta.

The true and false aneurysm occurs on any part of the abdominal aorta, but it is most frequently found above the cœliac axis, rarer at the cœliac axis, and rarer still at the bifurcation of the abdominal aorta; the dissecting aneurysm takes its origin mostly from the thoracic aorta near the heart, and it commences, therefore, above the abdominal aorta, and extends to a variable distance, forming a tube which mostly stops at the bifurcation, but may extend to the iliacs; the arterio-venous aneurysm, of which one typical case has been described by Pitt, is mostly situated at the bifurcation where the abdominal aorta is in close contact with the iliac veins.

As a rule, there is only one aneurysm, but occasionally a large one and several small ones have been found; the coexistence of thoracic and abdominal aneurysm has also been noticed.

The *anatomy* and *pathogenesis* of abdominal aneurysm is the same as that of aneurysm in other regions. The coverings of the false aneurysm vary according to its situation and direction of growth—when growing backwards the retro-peritoneal tissue, the vertebræ which are eroded, etc., form part of the covering, when growing upwards the diaphragm, when growing downwards the psoas muscle, if it grows in a forward direction the peritoneum covering the stomach, transverse colon or descending colon, or kidney may form part of its boundaries.

SYMPTOMS.—The symptoms vary considerably according to the rapidity and direction of the growth of the aneurysm. In some cases it may give rise to no symptoms, and sudden death may occur from rupture into the abdominal cavity; in others the symptoms may simulate spinal caries with compression-myelitis; in others the symptoms may simulate a new growth; in many cases, however, though the initial symptoms observed may be indifferent the aneurysm after a time gives rise to characteristic symptoms.

The symptoms may be divided into—

1. The physical signs of the aneurysm.
2. Pressure symptoms.
3. Secondary and general symptoms due to constitutional disturbance.

1. *Physical Signs.*—A *tumour* is noticed situated along some part of the course of the abdominal aorta, mostly in the epigastrium, or to the left of the epigastrium, rarely to the right, or the tumour may be detected in the back of the lumbar region forming a diffuse swelling. The tumour is smooth, more or less globular, and sometimes of an elastic feeling, somewhat compressible and often painful on pressure, and a distinct systolic thrill may be felt; the tumour may be fixed or movable, it may extend upwards and cause projection of the lowest ribs on the left side, or extend into the pelvis. The tumour does not move on respiration, or slightly so when adherent to the diaphragm or passing under the ribs. The percussion note over the tumour if it be large is dull, if it be small the stomach and colon overlying it may give a high tympanitic note. A most

important sign is *pulsation*, which may be visible, and if not visible it can be made out on palpation and on pressure; the pulsation is systolic or post-systolic, sometimes diastolic, and on placing the tumour between the hands, is felt to be distinctly expansile. On auscultation one often hears a systolic murmur over the aneurysm and sometimes beyond it, at other times only a systolic sound is heard, very rarely a systolic and diastolic bruit. Pressure and posture may alter the bruit, and may make it disappear. Occasionally the systolic murmur may be heard in the back, especially when visible bulging is noticed in the back, and occasionally, even, when only pressure symptoms occur and there is no tumour to be felt, this murmur over the back may be heard. On the other hand, it must be stated that the murmur may be absent, and that a murmur may be heard over a normal aorta by pressure of the stethoscope, and over a solid tumour resting on the abdominal aorta. In some cases the aneurysmal tumour is solid, not pulsating, and in this case a murmur may not be heard.

Other signs depending on the aneurysmal tumour are:—

(1) The pulsation does not disappear when the patient rests on his hands and knees.

(2) The pulse in the femorals is often retarded.

(3) Sudden pressure on the tumour causes a markedly increased tension in the femorals, and the sudden relaxation of the pressure diminishes the tension, and may even cause a disappearance of two or three beats in the femoral.

(4) Compression of both femorals causes an increased pressure in the aneurysm (a dangerous experiment, which might cause rupture of the aneurysm). In some cases the tumour shows no pulsation, and may give rise only to dulness on percussion and cause displacement of the viscera; occasionally the tumour may be very large and occupy the greater part of the left half of the abdomen, and in other cases, again, no definite tumour can be made out, but only a fulness on the left side which may give rise to a dull note on percussion, and extend from the seventh intercostal space to the crest of the ilium and laterally from the axillary line to the middle of spine.

2. *Pressure Symptoms, either Direct or Indirect.*—(1) *Pain* is the earliest and most constant symptom. It often exists long before the patient seeks advice, and generally persists to the last. It varies much in *character*, and it is often of neuralgic character, occasionally very severe; it may occur in paroxysms and resemble "colicky pain," closely simulating renal colic. The localisation and extension of the pain vary with the situation and growth of the aneurysm. The pain may be situated in the back over the spine and be of a gnawing character; if aneurysm is situated in the upper part of the aorta, the pain extends to lower ribs, or epigastrium: often the pain is in the lumbar region; it may by pressure on the lumbar or sacral plexus cause neuralgic pains along the course of nerves, and pain in the thigh, knee, and toe are not uncommon in abdominal aneurysm; the pain sometimes affects the testicle and penis. In some cases of false aneurysm, where the blood infiltrates the psoas muscle, pelvic pain is complained of. The pain is either the referred pain, as seen in other visceral diseases, or due to pressure on the nerves, at other times due to displacement of the organs by the growing aneurysm.

Other pressure symptoms are:—

(2) Weakness in the legs which may amount to complete paraplegia—this symptom may be due to the aneurysm causing erosion of the vertebræ, and pressing on the cord, or to obstruction of the abdominal aorta, which

either by causing anæmia of the spinal cord, or more likely, as shown by recent experimental investigation, by cutting off the blood-supply from the muscles and nerves, may produce a paralysis simulating transverse myelitis.

(3) Dyspnœa, due to upward extension of the aneurysm compressing the lungs. If the aneurysm is large the lungs may be very much compressed and the physical signs on examination may simulate pleural effusion. It must be noted that in some cases of abdominal aneurysm hydrothorax of the left side has been noticed, and this probably due to obstruction of veins and lymphatics. Persistent cough and extensive bronchitis have also been frequently noticed.

3. Common symptoms relating to the stomach and intestines are vomiting, flatulence, constipation; hæmatemesis may occur in rupture of the aneurysm into the stomach or duodenum, or it may be due to embolism in the arteries; constipation is a common symptom, pain in the rectum and in rarer cases melæna (rupture or obstruction to or embolism in the superior mesenteric artery). Dysphagia has been noticed in a few cases in which aneurysm grew upwards or compressed cardiac end of stomach or œsophagus.

4. The kidneys may be displaced downwards or latterly and be pressed upon. This may give rise to albuminuria (venous congestion); hæmaturia (renal infarcts or compression of renal artery); suppression of urine has been noticed in some cases in which the abdominal aorta above the renal artery was more or less obstructed by the aneurysm.

If the ureter be pressed upon or dragged by the displaced kidney, hydronephrosis may result. Cystitis and painful micturition have occasionally been observed.

5. Pain in the testicle occurring in paroxysms has already been noticed. In some rarer cases atrophy of one testicle (obstruction of spermatic artery) has been noticed (Walshe).

6. Pressure on the iliac vein may cause œdema of one lower extremity, and this was also noticed in an arterio-venous aneurysm (communication between iliac vein and abdominal aorta); pressure on the vena cava inferior causes œdema of both legs, and with or without œdema of the abdominal walls and ascites.

7. In females pressure symptoms may also be noticed in connection with the uterus and its adnexa.

8. Other symptoms in abdominal aneurysms difficult to explain are inequality of the pupils and also spinal myosis. This, I think, is more likely to be due to syphilis, which we know forms an important factor in the etiology of aneurysm. Hoarseness and aphasia have been noticed. The cause of this has not been clearly made out, and in one case in which these symptoms were noticed no affection of the recurrent laryngeal and nothing abnormal in the larynx was to be found at the autopsy (Hamilton Deekins).

Apart from these symptoms one notices in abdominal aneurysm secondary symptoms, such as palpitation, occasionally quick pulse, signs of dilatation or hypertrophy of the heart, not due to the aneurysm, but due to arterio-sclerosis, paroxysmal or persistent cough, often rise of temperature (perhaps due to the action of blood-ferment in the false aneurysm); owing to the excessive gnawing pain, which occurs sometimes when the vertebræ are eroded, the patient cannot move and holds his spine stiffly. The pain due to the pressure on the nerves may cause permanent contraction of the muscles; the patient often loses flesh to a considerable extent and has a pale cachectic appearance; this, however, often occurs only late in the disease.

As syphilis and arterio-sclerosis are often etiological factors in the causation of an aneurysm, symptoms relating to them may be found, such as vertigo, headache occurring at night, mental symptoms, such as melancholia, etc.

Course and Terminations.—The course of abdominal aneurysm is generally a very chronic one. In traumatic cases, in which we can often trace the beginning, the patient sometimes for a considerable time complains only of pain, increased by exertion, or of general weakness, especially at night; in the non-traumatic cases, similar symptoms arise, and the patient seeks only medical advice when a pulsating tumour is to be felt, of which the patient is himself often conscious, complaining of a throbbing sensation. In other cases the symptoms may simulate for some time those of a malignant tumour (of stomach, kidney, etc.). In a number of recorded cases the aneurysm gave rise to no special symptom till suddenly rupture occurred, or till symptoms of paralysis set in; when rupture occurs, one notices sudden collapse, great pallor, profuse clammy perspiration, and a thready pulse; this train of symptoms is soon followed by death, or the more urgent symptoms may subside, and the patient improves and lives for some time, when a second and fatal rupture occurs. Evidence of several ruptures of various dates has thus been found in not a few cases. Most cases of abdominal aneurysm terminate fatally. Death is most commonly due to rupture, which may take place into pleura, bronchi, mediastinum, pericardium, into the peritoneum, or retro-peritoneal tissue, when it may infiltrate various tissues, such as psoas muscle, and burrow along, and in one case the blood found its way through a sloughing bed-sore to the external surface; or the aneurysm may rupture into the stomach, duodenum, colon, pelvis of the kidney, bladder, vena cava, spinal canal, or gall-bladder. The rupture in some of these organs, such as the duodenum, pelvis of the kidney, may not prove immediately fatal, but may produce hæmatemesis and hæmaturia respectively; without rupture death may take place from general exhaustion, jaundice, obstruction of the aorta, or of superior mesenteric artery.

In some few cases, cure has been spontaneous; in other cases, treatment has brought about a complete cure; in many cases the patient improves for a time, but eventually death from rupture occurs.

Differential Diagnosis.—Here we may distinguish between (1) cases in which there is pulsating abdominal tumour; (2) cases in which there is an abdominal non-pulsating tumour; (3) cases where the aneurysm extends upwards, simulating aneurysm of thoracic aorta; or (4) cases in which paraplegia forms the principal symptom.

Ad 1. There is no difficulty in diagnosing the aneurysm from epigastric pulsation of a *dilated heart;* to distinguish aneurysm from a *pulsating abdominal aorta*, as a rule, is easy enough when we bear in mind that the pulsating aorta is found more often in women generally affected with neurasthenic symptoms, that the pulsations can often be followed to the bifurcation of the aorta, and that it is not expansile, it may disappear sometimes, and especially if the patient be examined under chloroform. A *solid tumour pressing* on the abdominal aorta is, in most cases, easily differentiated, for the tumour can often be lifted off the aorta, or it can be moved away (cases of pyloric cancer, movable kidney), or other secondary tumours are found, as in tumours of the omentum, of the retro-peritoneal glands; or some of the abdominal organs are found enlarged—liver, spleen (leucocythæmia, Hodgkin's disease). In some cases the diagnosis may be very difficult or even impossible.

One case I may mention in which the patient complained of excruciating abdominal pain localised over the epigastrium, and also of pain over the lowest dorsal vertebra; the patient emaciated rapidly; no tumour could be felt, but there was a loud systolic bruit over the left lumbar region; the patient had profuse diarrhœa, but the stool, which was examined often, as the case was thought to be one of tubercular enteritis, showed no increase of fat. At the autopsy, cancer of the tail of the pancreas encircling the aorta was found.

If the tumour extends upwards, it may simulate pulsating empyema; if the examination of the heart and the absence of the other signs of empyema should not give sufficient data, the application of the Röntgen rays should clear up any difficulty. This new method of physical examination would also help us to distinguish abdominal aneurysm from aneurysm of the *descending thoracic* aorta by watching the movements of the diaphragm in relation to the tumour.

If the pulsation is noticed on the back, a systolic bruit is most constantly heard, and this would help us to distinguish it from *perinephritic abscess* or *abscess from caries of the vertebræ* along with other symptoms, such as redness of the skin, pyrexia, etc.

Ad 2. In cases in which the tumour is not pulsating, the distinction from fæcal accumulation is not difficult (doughy inelastic feel, position of the mass, history of the case); from malignant disease of the kidney or colon it may be more difficult, also if it extends downwards it may simulate tumour springing from the pelvic organs or arising from the walls of the pelvis—regard must be had to the history of the case, the presence, character, and distribution of the pain, and, as in most of the published cases of this form of aneurysm the vertebræ were found much eroded, the skiagraph would form an important aid in the diagnosis; also the retardation of the pulse in the femorals.

Ad 3. When the tumour extends upwards and does not give rise to pulsation it may simulate an *empyema*. Besides the history of the case, a careful examination of the chest ought to clear up any doubt, for a systolic bruit is often heard, and the heart is pushed upwards or (as in one case which I saw) is found pulsating forcibly against the chest wall by being pushed forward. The distinction between *thoracic* and abdominal aneurysm has already been noticed.

Ad 4. The diagnosis of those cases in which paralysis of the legs sets in, and in which the affection simulates caries, transverse myelitis, or a tumour of the vertebræ, has already been dwelt upon. If the paralysis is due to obstruction of the aorta, no pulse will be felt in either femoral; if it be due to pressure on the cord, the Röntgen rays ought to clear up any doubt, as it would show the simple erosion, uncomplicated by the inflammatory changes in the bone seen in caries.

When rupture has taken place, the diagnosis is not difficult, but the collapsed condition has to be distinguished from *extra-uterine fœtation, perforative peritonitis, acute pancreatitis.*

Early diagnosis of abdominal aneurysm is a matter of importance, and incurable lumbago and sciatica require the most careful physical examination with all modern accessories, sphygmograph, skiagraph.

Dissecting aneurysm often gives rise to no symptoms, and is discovered at the autopsy or gives rise to such indefinite symptoms that, unless there are signs of obstruction of the aorta or its branches, the affection cannot be diagnosed. It generally causes death from rupture of the aorta.

Treatment of Abdominal Aneurysm.—I. Medicinal.—It is needless to refer to this, as it is the same as for thoracic aneurysm, to the article on which I must refer. Of recent additions on this subject I may mention (1) the treatment by the administration of chloride of calcium. This, according to Professor Wright's observation, increases the coagulability of the blood, and appears to have been successfully used in two cases of abdominal aneurysm.

(2) The subcutaneous injection of gelatin, which has been first tried by Lancereaux and Paulesco (*Bulletin de l'Académie de Med.* 1897, vol. xxxvii.; 1898, vol. xl.), who found that the subcutaneous injection into the gluteal region of about 200-2·250 cbc. of a sterilised 3 per cent watery solution of gelatin causes rapid consolidation and eventual cure of the aneurysm. The method has since been tried with varying success by several German and French observers (*Deutsch. med. Wochen.* 1899). Lancereaux found experimentally in animals that when gelatin was introduced into the system the blood coagulated much quicker. Two cases are recorded in which death occurred soon after gelatin injection (Barth, *Gaz. des hôpit.* 1898). (3) The administration of proto-nuclein is said to have caused the cure of an abdominal aneurysm (*New York Med. Journal*, Sept. 1898).

II. Compression of the abdominal aorta. (1) Pressure on the proximal side, as by far the largest number of aneurysms are found above the cœliac axis; this method can only be applied in a few cases. The first and best-known case in which this was successfully tried is Murray's (*Med.-Chir. Trans.* 1864). H. Moxon and Durham (*Med.-Chir. Trans.* 1872) have published a second successful case, and since then very few cases have been published. The pressure is applied for several hours at a time at different intervals. Consolidation, according to Murray, is set up very quickly.

(2) Pressure on the distal side. Pary and Bryant (*Med.-Chir. Trans.* 1872) record a case in which Lister's tourniquet was applied for twelve hours, and again for four hours after an interval of twelve hours. The patient died soon after from peritonitis.

(3) Ligature of the abdominal aorta, proposed here by Astley Cooper, has not found any advocates in recent years.

III. Introduction of foreign bodies. This applies to thoracic as well as abdominal aneurysm; needles, horse-hair, iron wire, platinum wire, steel springs, have been used in abdominal aneurysm. In some cases excellent results obtain (see Boyd, *Brit. Med. Journ.* 1899, vol. i.). M'Ewen's method for the production of a white thrombus applied equally to both forms of aneurysm.

IV. Injection of blood-coagulating substances (perchloride of iron, alcohol, ergot, tannin) is only applicable in exceptional cases, and of very doubtful use (Smith, *Barth. Hospit. Reports*, 1891).

V. Electrolysis is not applicable for abdominal aneurysm.

Aneurysm of the Branches of the Abdominal Aorta

Cœliac Axis.—It is mostly the aorta itself, close to the cœliac axis, which gives rise to the aneurysm, and only in a few cases does the aneurysm spring from the axis itself. In its symptoms, course, and termination it does not differ from aneurysm of abdominal aorta.

Gastric Artery.—Of very rare occurrence, occasionally the aneurysm springs from the gastric artery near its origin; more interesting are those cases (Douglas Powell, *Path. Trans.* 1878; Gerry, *Boston Med. and Surg. Journ.* 1888) in which the aneurysm formed at the bottom of a gastric ulcer and caused death by hæmorrhage into the stomach, or into the peritoneal cavity (Cuming, *Dublin Quart. Journ. of Med.* 1869, vol. xlvii.; Villard, *Bull. soc. anat. de Paris,* 1870, xiv.).

Splenic Artery.—The aneurysms of the splenic artery form a peculiar group, inasmuch as one finds several, and as, in most recorded cases, the aneurysm, about the size of an egg or orange, had a hard, calcareous covering and communicated only by means of a small opening with the artery. The contents of the sac are soft, putty-like, like the contents of a sebaceous tumour. For this form of aneurysm Corvisart designed the name of cystogenous aneurysm.

These aneurysms are small, they give rise to few symptoms (Turner, *Path. Trans.* vol. xxxvi. p. 151), and terminate by rupture into the peritoneal cavity or into splenic vein (Weigert, *Virchow's Arch.* vol. civ. 1886), or are found accidentally after death (Davidson, *Liverpool Med.-Chir. Journ.* 1884, p. 210). Goodhardt (*Path.* vol. xl. p. 67) describes a case of arterio-venous aneurysm which had caused thrombosis of the mesenteric vein and a localised colitis (*Lancet*, 1885, vol. i. p. 518); rupture took place into the stomach in Osler's case (*Montreal Gen. Hosp. Rep.* 1880, 1). A branch of the splenic artery, the gastro-epiploica sinistra, has in rare cases been found the seat of aneurysm in connection with a gastric ulcer.

Hepatic Artery.—Above twenty cases of this affection have been recorded. The aneurysm may reach a large size (size of fist or more); it is either situated between liver and small curvature of stomach, occasionally it forms a tumour in the situation of the gall-bladder, in other cases it was found on the under surface of the liver. Amongst the symptoms it gives rise to we may mention—pain of a severe character, often paroxysmal, jaundice, and hæmorrhage in the form of melæna or hæmatemesis, which may go on for some time. The aneurysm, which in its symptoms may simulate gall-stones or duodenal ulcer, finally ruptures either into the peritoneal cavity, or duodenum, or gall-bladder, or into the biliary duct, or it may give rise to multiple abscesses in the liver.

For literature up to 1886 see Caton (*Clin. Soc. Journal*, 1886, p. 275); the more recent cases are recorded by Mester (*Zeitsch. f. klin. Med.* 1895, vol. xxviii.); Schmidt (*Deutsch. Arch. f. klin. Med.* 1893-94, vol. lii.).

Mesenteric Arteries.—The superior is much more often affected than the inferior. (I find a record of twenty cases of the former and of five of the latter.) Several of the cases recorded are due to embolism; and the principal symptom noticed has been hæmorrhage from the bowels; in Gairdner's case (*Month. Jour. of Med. Science*, London and Edinburgh, 1850) the aneurysm ruptured into the duodenum, and caused hæmatemesis from time to time, thus simulating gastric ulcer; the patient died many months after from rupture of the aneurysm into the peritoneal cavity; B. Yeo (*Path. Trans.* 1877, vol. xxviii. p. 94) describes a case in which both renal arteries were compressed, and the patient died from renal coma. Other cases are given by Ogle, *Lancet*, 1857, ii. 87; Gull, *ibid.* 1859, v. 484; Wilks, *Path. Trans.* 1859-60, xi. p. 44; Galloway, *Path. Trans.* 1890-91, xlii. p. 78. Of aneurysm of the inferior mesenteric artery we have records by Peacock, *Path. Trans.* 1861, xii. p. 73; Rodet (ulcer, ulcerative endocarditis), *Mém. Soc. de Sc. méd. de Lyon*, 1864, iii. 155; Cockle, *Lancet*, 1864, vol. i. No. 18.

Renal Artery.—I find twelve recorded cases of aneurysm of renal artery (small aneurysms, according to Osler, are not uncommon). They are diffuse, and a tumour could in some cases be made out, but not always pulsation. Death occurred from rupture either into the peritoneal cavity or retro-peritoneal tissue, or into the pelvis of the kidney, producing hæmaturia. Hahn (*Deutsch. med. Wochensch.* 1894, No. 32) describes a case of aneurysm of a branch of the renal artery which simulated cancer of the kidney, and in which removal of the kidney brought about complete recovery. Several of the recorded cases (Gruber, *Wien. med. Wochensch.* 1891, xli. p. 1646; Turner, *Path. Trans.* 1884-85, xxxvi. 277; Illich, *Riforma med.* vii. pt. 2, p. 481, etc.) were of traumatic origin.

SPERMATIC ARTERY (see Oliver, *Cincin. Lancet*, 1893, N.S. xxx. 197).

PHRENIC ARTERY.—Only one case recorded. Dean, "Aneurysm of the Left Phrenic Artery," *St. Louis Med. and S. Journal*, 1884, xlvi. p. 47.

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Abdominal Operations, Technique of. *See* LAPAROTOMY.

Abdominal Tumours, Diagnosis of.

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IT is not an easy matter to decide as to the proper limits of the term "abdominal tumour." Pathologically the word "tumour" is used in reference to a proliferating growth or neoplasm, but clinically it is convenient, at any rate in the case of the abdomen, to use the word in a wider sense, applying it not only to a neoplasm, but to an abscess, to a cyst, and even to a simple enlargement of an organ. Thus a dilated stomach, a hydronephrosis, an abscess of the liver, and a floating kidney are often spoken of as abdominal tumours.

The correct diagnosis of a tumour of the abdomen implies a knowledge (1) of its site—that is, the organ or structure from which it grows or of which it is a part; and (2) of its pathological nature. This knowledge is only acquired by a careful study of the clinical history and pathology of the various lesions that affect the walls of the abdomen and its contents; it will be set forth in future articles dealing with these subjects. At present it must suffice to indicate some of the guiding lines which are of assistance in diagnosis and to group together some of the chief features presented by tumours involving special organs or tissues.

A few points of general importance in connection with the subject may first be noticed. In the first place, a tumour does not always produce a localised swelling, it may be big enough to cause a general and uniform enlargement of the abdomen; this happens, for example, in some cases of ovarian cysts, retro-peritoneal growths, enlarged spleen, etc. In the second place, the situation of a tumour does not necessarily correspond to the normal position of the organ or tissue involved by it; thus a small hard mass below the left costal arch may be due to cancer of the pylorus, a cylindrical swelling in the left flank to an ileo-cæcal intussusception, and a large tumour in the left lumbar and iliac regions to a uterine fibroid or ovarian tumour. Then, again, the possibility that an abdominal swelling is merely caused by an accumulation of fæces should always be considered; hence it is necessary to thoroughly empty the bowels before coming to a conclusion with regard to any abdominal tumour. Similarly, in doubtful cases, the possibility of the tumour being due to a distended bladder must be borne in mind.

Thirdly, the existence of pulsation must be investigated. An aneurysm of the abdominal aorta produces a more or less defined pulsating tumour which with each pulsation will separate the fingers applied to its sides. Its site is usually above and to the left of the umbilicus, but it may take up any position on the left side, and even rarely be present on the right side.

Increased aortic pulsation is frequently met with in anæmic subjects, but this is not expansile, and lateral pulsation is not perceptible. It is a good practical rule to *exclude aneurysm* when the presence of expansile pulsation is doubtful. New growths often receive an impulse from the aorta, but they rarely present lateral pulsation; and when the patient rests on his elbows and knees the tumour falls away from the aorta, and its pulsation ceases to be felt.

Lastly, the influence of the respiratory movements requires consideration. Any tumour which is connected directly or indirectly with the diaphragm may be felt to rise and fall during inspiration and expiration. Thus tumours of the liver and spleen descend when a deep inspiration is taken, while tumours of the omentum and bowels are commonly uninfluenced by it.

In taking a survey of some of the chief features of abdominal tumours it is convenient to consider first tumours affecting the abdominal parietes, and, secondly, tumours occurring within the abdominal cavity itself.

TUMOURS OF THE PARIETES.—These may be caused by an excessive deposit of fat, by abscesses, by muscular contractions, and by new growths. The last-mentioned are rare, and when present are usually secondary to a neoplasm within the abdomen; thus a cancerous growth about the umbilicus occasionally occurs when there is cancer of the stomach or omentum.

A tumour of the parietes is usually recognised by its superficial position; it is also more apparent when the patient raises himself from the bed without the assistance of his hands, and is more easily felt when he assumes the knee-elbow position.

With regard to swellings caused by muscular contraction the possible coexistence of visceral disease must not be overlooked. The so-called "phantom tumours"—referred to in a previous article—which are sometimes associated with an abnormal deposit of fat in the abdominal wall or in the omentum, are occasionally merely manifestations of hysteria, but very often they are of reflex origin and depend on some inflammatory process such as pelvic peritonitis or other internal disease. Hence it is of the highest importance in such cases to *make a complete examination of both the pelvic and abdominal organs.*

TUMOURS ARISING WITHIN THE ABDOMEN.—*The Liver.*—In health, although the liver stretches across the upper portion of the abdomen, its lower edge is but rarely palpable, and it may be laid down as a reliable rule in practice that when the lower edge can be distinctly felt the organ is either displaced downwards or enlarged.

The characteristic features of a swelling due to or connected with the liver are—(1) that it occupies mainly the right hypochondriac and epigastric regions; (2) that it may be felt to descend during a forcible inspiration; and (3) that the dulness, which is usually obtained by percussing over it, is continuous with that obtained over the hepatic costal region.

Leaving out of consideration the various kinds of simple enlargement, such as those produced by cirrhosis or fatty degeneration, the usual causes of a prominent mass or swelling are—*cancer, abscess, syphilis,* and *hydatids.*

Abscesses or hydatid cysts often produce a tense, round, smooth swelling, which usually implicates the right lobe of the liver, but the whole organ may be greatly enlarged and extend below the umbilicus. Sometimes the enlargement is chiefly behind, and may ascend high into the pleural cavity; there is also frequently a prominent bulging of the lower portion of the right side of the thorax. A liver abscess is commonly tender to pressure, whereas a hydatid cyst often runs a painless course. Fluctuation or an elastic sensation, according to the depth from the surface, may be obtained over both abscess and cyst. A peculiar tremulous sensation, known as "hydatid thrill," may be conveyed to the left hand laid flat on an echinococcus tumour, when the latter is percussed with the fingers of the right hand. This thrill, however, is met with over some ovarian cysts and rarely in cases of ascites.

Gummata produce flat or nodular projections of various sizes, and may be associated with cicatricial contractions on the surface of the liver, constituting, in some cases, an irregular nodular mass in the right hypochondrium or epigastrium.

Cancer, which is usually a secondary growth, gives rise to great enlargement of the liver and also to large outgrowths on the surface or at the edge of the liver. These have rounded margins and cup-shaped central depressions. A cancerous growth may be simulated by the irregular syphilitic liver, but the former undergoes progressive enlargement and is also distinguished by its association with profound cachexia, emaciation, and jaundice.

The Gall-Bladder.—A distended gall-bladder gives rise to a superficial, pyriform or globular, tense, elastic swelling which projects beneath the costal margin between the right nipple and parasternal lines. Below, its outlines can be defined; but above, no definite edge can be felt. The swelling may depend on obstruction of the cystic duct by calculi or cancer. Rapid emaciation, with or without jaundice, and cachexia, speak for cancer. When the fundus of the bladder is cancerous, the swelling is hard and perhaps nodular and is less movable than in simple hydrops.

The Stomach.—Two varieties of abdominal swelling occur—one usually small and hard, due to new growth, and the other large and elastic, due to distension of the stomach. The former tumour is usually felt above and to the right of the umbilicus and is due to cancer of the pylorus, for this part is easily dragged down from its normal position at the right costal margin. The mass is usually circumscribed, except on the left, where sometimes it may be felt to shade off gradually into the wall of the stomach. It may or may not be affected by the respiratory movements.

In a suspected case of cancer of the pylorus, when there is no obvious prominence, the observer's fingers should be pushed as deeply as possible beneath the edge of the right rectus muscle, while the patient lies on his back or on one or other side. In this way a small nodule may be discovered which otherwise would have escaped notice. It is also to be observed—(1) that a tumour may displace the pylorus to a considerable extent, thus cancer of this part may be situated as far to the left as the hypochondrium, and rarely even below the level of the umbilicus; (2) that the pulsation of the aorta may be transmitted through the tumour; (3) that such pulsation, and even the tumour itself, may be felt to-day and gone to-morrow; (4) that gurgling of gas may be felt through a pyloric tumour.

A tumour in the epigastrium and left hypochondrium occurs in cancer of the body of the stomach; also in such rare conditions as diffuse sarcoma and chronic induration of the wall of the stomach.

Dilatation of the stomach, the usual cause of which is pyloric obstruction, may cause distension of any part of the abdomen, except perhaps the epigastrium. Frequently in chronic dilatation the stomach occupies a lower position than normal; and its contour is plainly seen, chiefly owing to the presence of peristaltic waves which pass from left to right; they are often associated with irregular protrusions of the stomach walls.

The Intestines.—A portion of the bowel may be found in a state of distension, or it may be the seat of a tumour. The latter is commonly produced by one of four causes, viz. fæcal accumulation, inflammatory exudation, new growths, intussusception. A *fæcal tumour*, which may occur in almost any part of the abdomen, is usually cylindrical in shape, has an uneven surface, and feels as if it were situated immediately beneath the abdominal wall. *Inflammatory swellings* are more commonly situated in the right or left iliac fossa—a swelling on the right side being usually due to appendicitis. These swellings are associated with pain, tenderness, and symptoms of febrile disturbance. Abscesses may form and point in various directions. *Cancer* of the cæcum produces a harder and more nodular mass than that due to appendicitis, and is usually a little nearer to Poupart's ligament. If it causes obstruction of the bowel the flanks may be flattened when the rest of the abdomen is much enlarged.

Rarely a swelling in the right hypochondrium is caused by cancer of the duodenum or of the hepatic flexure of the colon; one in the left hypochondrium by cancer of the splenic flexure. But the commonest site for cancer of the bowel is the rectum or sigmoid flexure. In malignant disease of the hepatic, splenic, or sigmoid flexure the symptoms are often referred back to the cæcum, which is liable to become distended and ulcerated. Bubbling of gas may be felt through these tumours, and peristaltic waves passing from right to left may be seen to travel along the distended portion of colon. *In intussusception* there is a sausage-shaped tumour in the course of the colon which hardens during manipulation or during attacks of griping pain.

The Spleen.—As a rule an enlarged spleen is easily felt; it projects as an oval solid tumour from below the left costal arch, and passes downwards towards the iliac crest and forwards towards the middle line. It is superficial and mobile, and can be felt to descend with each inspiration. The upper or anterior margin is usually thin, and presents one or two well-defined notches. The lower or posterior edge feels rounded, and is quite free. The greatest enlargement occurs in leucocythæmia, in which disease the spleen may extend into the right side and cause a general enlargement of the abdomen. Abscesses, cysts, and new growths of the spleen are rare. They produce swellings or projections from the surface of the organ. A movable spleen is sometimes present, and, in extreme cases, may occupy any region of the abdomen. It has been mistaken for a fibroid or an ovarian tumour.

The Pancreas.—The diagnosis of a tumour of this organ is often very difficult. The stomach and colon should be empty, and the abdominal walls rendered as lax as possible. Cancer of the head of the pancreas sometimes gives rise to a tumour situated somewhere between the umbilicus and the ensiform cartilage and usually to the right of the middle line. The tumour is fixed, deep-seated, hard and nodular, and does not move with respiration. The gall-bladder is often distended; there is rapid emaciation and often intense and persistent jaundice. A cyst of the pancreas often reaches a large size, and forms a rounded fluctuating tumour in the upper part of the abdomen; rarely a cyst has developed in the tail of the pancreas and produced a swelling in the left hypochondrium.

It must be remembered that a swelling in the epigastric, umbilical, and left hypochondriac regions may be due to hæmorrhagic or other fluid accumulations in the *lesser peritoneal cavity.*

The Omentum.—Tumours of the omentum are felt as irregular lobular masses in the lower epigastric and umbilical regions. They are superficial, usually dull to percussion, and rarely descend a little during inspiration. Tubercular deposits may form lumps above the umbilicus or sausage-shaped tumours which stretch across the abdomen. Cancer of the omentum may simulate cancer of the liver or stomach; from the former it may be distinguished by a band of resonance between it and the liver, from the latter by a careful examination of the stomach, both when empty and when distended by inflation.

The Mesenteric and Retro-Peritoneal Glands.—Tubercular or cancerous deposits may produce palpable firm masses which are more or less fixed, and are situated at or below the level of the umbilicus. In some cases a glandular mass transmits pulsation from the underlying aorta. Examination per rectum may detect cancerous glandular masses pressing down on its upper wall, and leading sometimes to partial obstruction of the bowel, and ascites may coexist from cancerous disease of the peritoneum.

The Kidney.—In some subjects, especially in children and women with lax abdominal walls, a healthy kidney may be felt by bimanual palpation, but as a rule a kidney that can be distinctly felt is either displaced or enlarged. A displaced—"movable" or "floating"—kidney may be felt as a smooth, slippery tumour in the lumbar or umbilical region, and rarely even in other parts of the abdomen. It may be pushed in almost any direction, but more easily upwards than downwards. In women, in whom it occurs most frequently, it is liable to become enlarged and more tender to palpation during the menstrual period. Sometimes the enlargement is due to hydronephrosis, rarely to cancer.

In a large number of cases of movable kidney there are no symptoms; in others there are neurasthenic and dyspeptic manifestations; while in a third series of cases the patient is subject to feverish attacks in which there are severe abdominal pains, shivering, and vomiting, and which may lead to considerable emaciation.

A tumour of the kidney—the result of hydronephrosis, malignant disease, a cyst, or an abscess—fills up the loin and extends forwards towards the front of the abdomen. It rarely causes enlargement behind, and may thus be distinguished from abscesses and other lesions outside the kidney, which frequently cause much projection posteriorly.

A renal tumour on the left side may resemble an enlarged spleen; a hollow may usually be felt between an enlarged kidney and the costal margin, whereas an enlarged spleen passes beneath the ribs. In front, too, of a renal tumour the colon is commonly felt as a soft or yielding structure, sometimes as a thickened cord. Inflation of the colon is frequently of aid in diagnosis. The anterior margin of an enlarged kidney is rounded, whereas that of an enlarged spleen is sharp and notched. A distended gall-bladder may resemble a fluctuating swelling of the right kidney; but the latter can be traced back towards the spine, whereas the former is superficial and rises and falls with the respiratory movements.

The Uterus.—A uterine myoma, when large enough to rise out of the pelvis, produces a firm mass in the middle line; it is dull to percussion, and sometimes a loud systolic hum is detected by auscultation. *The diagnosis* has to be made from a distended bladder from an ovarian tumour, and from a uterus enlarged from causes other than fibroid tumour, such as pregnancy, retention of menstrual fluid, etc.

The Ovaries.—An ovarian cyst also occupies, as a rule, the middle line, though there may be a history of its commencement on one or other side. It produces a swelling of the abdomen which is most marked below the umbilicus. There is dulness in front and resonance in the flanks, the reverse being the case in ascites. The uterus is found to be of normal size. Sometimes a cystic tumour of the broad ligament simulates an ovarian tumour.

The *Fallopian tubes* may also be the seat of tumours, such as malignant and tubercular growths, which can be felt through the abdomen; or they may become enormously distended by accumulations of clear fluid, pus, or blood.

The possibility of an advanced extra-uterine pregnancy must also be borne in mind.

Abortion.

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DEFINITION.—Abortion or Miscarriage (French *Avortement,* German *Fehlgeburt*) indicates interruption of utero-gestation before the fœtus is viable, as distinguished from the interruptions that occur during the last trimester of pregnancy when the infant expelled is viable. Sometimes the term "abortion" is restricted to the interruptions that occur before the date of quickening, and the designation "miscarriage" is applied to those that occur between the fourth and the seventh months of gestation. All cases, however, where the ovum is addled or expelled before the sixth month are to be regarded as abortions. The abortion may occur at any date, but is more frequent at the third month. Statistics (Whitehead) would seem to indicate that four-fifths of all abortions occur between the second and fourth months. The occurrence most frequently coincides with a menstrual epoch.

FREQUENCY.—It is not easy to determine the frequency with which abortion takes place. The difficulty of determining the relative frequency of aborted to completed pregnancies is greatly due to the inattention of patients to their menstrual functions. A period passes without any loss of blood, and when it escapes six, eight, or ten weeks later in larger quantity than usual and mixed with what they take to be clots, they imagine that it is only an overflow due to the delay, whereas in reality they had aborted. The following statistics may be given:—

	Abortion.		Full-time labours.
Hegar	1	in	8 or 10
Devilliers	1	in	3 or 4
Whitehead	1	in	7 (the average age being below thirty).

VARIETIES OF PRODUCT.—Jacquemier, without giving the reference, says Guillemot has proposed to group abortions according as the uterus casts its contents—(1) in the first three weeks, when the embryo is still a relatively small part of the ovum, and the abortion is to be designated OVULAR; (2) between that date and the third month, when the different parts of the fœtus are taking shape and the placenta is undergoing development, and the abortion to be called EMBRYONIC; (3) from the third month onward, after the infantile form is fairly assumed, and the abortion is to be spoken of as FŒTAL. But the mole or mass that is expelled in a case of abortion does not necessarily represent the ordinary contents of the uterus at the patient's supposed period of pregnancy. While the embryo may have been dead for some time it may not be extruded, and parts of the ovum undergo further changes which prevent their identification with the date of this expulsion. Even where the products do correspond to the gestation date, the abortion does not go on in every case in the same way.

IN AN ABORTION AT THE THIRD MONTH, *e.g.* where under the regular action of the uterus there has been a perfect deciduation of the mucosa, the mole will be of the size of a large goose egg, and present on its outer surface the tomentous appearance of the external surface of the decidua vera, with the placental development in the serotinal area. This represents a complete abortion. But often the decidua vera remains attached to the uterus, and the ovum escapes invested only with the still vascular though less red reflexa and the placenta. In that case the general surface is smooth, with only the roughnesses of the placental portion. Sometimes the body that is passed has a white appearance, with one part shaggy or bringing serotinal tissue with it, whilst the rest of the surface is relatively smooth. In that case we have an ovum that has burst through all its involucra and comes off invested with the chorion. At other times the chorion has given way, and we see a sac with transparent wall which we recognise as the amnion, containing the fœtus floating in its liquor amnii. In other instances all the membranes have broken and the liquor amnii and fœtus may first escape, leaving the rest of the uterine contents to be expelled piecemeal or in more or less of their entirety.

WHEN THE EMBRYO DIES IN EARLY PREGNANCY, it sometimes disappears entirely, so that when the amniotic cavity is opened it will be found to contain nothing but liquid; or it may be that a fragment of umbilical cord is attached to the surface where the placenta had begun to form. Where it has not disintegrated it may be found of the size of a bean in a cavity large enough to have contained a three or four months' fœtus. If death set in at a later stage, and abortion do not immediately follow, the fœtus becomes macerated and shrunken, or its tissues and cavities become infiltrated and filled with a serous transudation which is often sanguinolent. More rarely the liquor amnii has become absorbed, so that the fœtus is crushed together; and when in a geminal pregnancy the twin in the upper part of the uterus perishes at the third or fourth month, it becomes flattened between its growing neighbour and the uterine wall, and appears at birth as a *fœtus papyraceus* or *fœtus compressus*. A twin ovum that dies in the lower segment of the uterus may on the other hand be discharged and have the appearance of an ordinary abortion, whilst the living twin may go on developing and be born as a full-time child.

Beside the diversities due to the varying conditions of the embryo and fœtus, the products of an abortion present further differences according to changes in the walls of the ovum or in their uterine investments. As a result we get a "mole"—either a "fleshy mole" (mola carnosa), when the discharged mass has the appearance of a piece of flesh, and consists of a cast of the whole uterus; "a bloody mole" (mola hæmatosa or cruenta), the result of extravasation into the tissues or cavities; or a "hydatid, vesicular, or bladdery mole" (mola vesicularis or hydatosa), resulting from the so-called myxomatous degeneration of the chorionic villi.

CAUSES

The causes of abortion have sometimes been set down to conditions on the part of the husband. Old age, weakness from sexual excesses, syphilis, phthisis, renal disease, and such like, may have left a man capable of fertilising an ovum, but to such an imperfect degree that it does not attain its full development. The paternal influence, however, is not the direct cause of the abortion, which must be sought in morbid changes either (1) in the ovum, (2) in the placenta, or (3) in the mother.

1. Ovular or Fœtal Causes.—The causes of the part of the ovum are found sometimes in the fœtus itself, but more frequently in the investing membranes. They may be classified as follows: (1) Death of the Fœtus; (2) Diseases of the Membranes. The various factors inducing death of the fœtus, and the morbid conditions of the investing membranes leading to abortion, are fully discussed later (*vide* "Pregnancy, Pathology of").

2. Placental Causes.—Intermediate between the causes that are referable on the one hand to the ovum, and on the other to the mother, are those which depend on morbid changes in the placenta, composed of elements derived from both. We may note the tendency to abortion due to abnormalities in—

(*a*) *The Placental Area.*—Hegar's observation has often been confirmed that where the area of attachment of the chorion to the uterus is unduly narrow, the ovum is easily detached and thrown off, and that on the other hand, where the serotinal attachment is of unusual extent, hæmorrhages are apt to arise which excite the uterus to abort.

(*b*) *The Placental Site.*—When the implantation of the placenta is abnormally low, hæmorrhages and abortion may occur in the earlier months. If gestation continues, its existence is revealed by accidental or unavoidable hæmorrhage.

(*c*) *Morbid Changes in the Placental Elements* (*vide* "Pregnancy, Pathology of").

3. Maternal Causes.—(1) *Local Causes.*—These are in the uterine membranes, in the uterus as a whole, or in the neighbouring organs.

(*a*) *Decidual Disease.*—A great many cases of abortion are traceable to inflammatory processes occurring in the endometrium, whether these had been present before impregnation began or set in more acutely afterwards, and often abortions associated with general diseases, such as cholera, result more immediately from the endometritis induced by the diseases.

(*b*) *Uterine Disorder.*—Again, if there be neoplasms such as fibroid tumours in the walls, or cancer developing in the neck of the uterus, abortion is liable to occur. So also pregnancy in a displaced and especially in a retroflexed uterus, or where there has been laceration of the cervix, very often ends abortively.

(*c*) *Disease of the Adnexa.*—Tumours of the ovary, distensions of the Fallopian tubes, and inflammatory adhesions with some of the neighbouring organs sometimes interfere with the normal growth of the uterus and cause abortion.

(2) *Constitutional Causes.*—(*a*) *Chronic Diseases.*—Where the system is weakened by over-lactation, by sexual excesses, alcoholism, or by want in times of famine, or from anæmia, chorea, diabetes, albuminuria or heart disease, or even habitual constipation, the inability to bear the strain of pregnancy shows itself in some by the occurrence of abortion and in others by the onset of premature labour.

(*b*) *Acute Diseases.*—In various acute diseases, such as the zymotic fevers, acute yellow atrophy, pneumonia, peritonitis, etc., gestation is apt to be cut short. Poisoning with lead or mercury, or with noxious vapours such as coal-gas or carbonic acid gas, acts in the same way.

(*c*) *Shock or Injury.*—In women of a nervous temperament a sudden fright, in others a physical jar or injury will cause abortion. The risk of abortion from surgical interference has been greatly lessened since the patient's fears are quieted by the administration of anæsthetics and the tendency to surgical fever counteracted by the use of antiseptics.

Criminal Abortion.—The practitioner must not lose sight of the possibility that he may be called to a case where he is unable to trace the abortion to any of the causes we have been considering. Even in unlikely quarters he may find that a woman has tried at her own hand, or with the advice and aid of an abortionist, to free herself of the fruit of an illicit amour. There is too sad evidence of a disposition among married women of frivolous character to crush the noblest instinct of womanhood and sacrifice the life that is palpitating in their womb, that they may be free to meet the claims they let society impose upon them. It is usually at the second or third month that such criminal procedures are had resort to.

Symptoms and Diagnosis

The Symptoms that lead the patient to send for the practitioner are—(1) Hæmorrhage; (2) Discharges from the Uterus; (3) Pain.

1. *Hæmorrhage.*—Very commonly it is the oozing of blood from the genital canal that gives the first signal of the danger. The bleeding may present the following characters:—A mere show, an ordinary menstrual period, or a profuse flooding. It may appear intermittently at intervals of hours or days, or in a more continuous stream, and may be fluid or clotted. Sometimes the blood has coagulated within the uterus, and only the serum is making its escape. In abortions from the fourth to the sixth month there may be no hæmorrhage till the birth-process is nearly over; but in the earlier abortion there is usually hæmorrhage from the outset. The possibility of the hæmorrhage arising from an extra-uterine gestation must not be lost sight of.

2. *Other discharges.*—If the abortion begin with the burst of the membranes, escape of the liquor amnii may be the first indication. Watery discharges may be due to decidual endometritis or be associated with hydatigenous degeneration of the chorion. In the latter case some of the vesicles may be detached and thrown off. In other instances some portions of the membranes, uterine or ovular, or the fœtus, may be discharged. Now and again the whole contents of the uterus are thrown off almost before there has been any loss of blood.

3. *Pain.*—The expulsion of the uterine contents is brought about by the action of the uterine muscles. Accordingly, intermittent pains, indicative of the uterine effort, and reminding a woman, who has previously had a child, of her labour pains, are perceived sooner or later. Sometimes it is the occurrence of the pains that makes her appreciate her danger.

Diagnosis.—The full diagnosis, however, can only be arrived at by direct physical examination. Perhaps inspection of a "mole" that has been discharged may convince the practitioner that he has before him the untimely product of a conception, and that the abortion is already complete. Even in that case it would still be his duty to make a bimanual examination of the uterus to make sure that it is contracting, is in good position, and not unduly hæmorrhagic. Where as yet nothing but some blood has escaped, he must examine to determine whether the patient be really pregnant, and whether the abortion be evitable or inevitable.

1. *Is she Pregnant?*—The differential diagnosis of pregnancy is discussed under "Pregnancy." The following points should be specially noted on vaginal examination. At the third month, the cervix of the gravid uterus is beginning to soften, whilst the body of the uterus is distinctly enlarged and

expanded above the neck, the anterior and posterior walls being separated from each other. The walls are notably softened, as can be felt most clearly through the posterior fornix, or better still, by the middle finger passed farther up the back wall of the uterus through the rectum. If the examination be made with some care, a change will be found to take place at times in the uterine walls, which harden as a wave of contraction passes through their muscular fibres.

2. *Is the Abortion inevitable?*—In trying to answer this question, three points fall to be noted. (1) *The character of the pains.* If these are weak, recurring at long intervals and controllable, the mischief may be averted; where, on the contrary, they are strong, frequently recurring and beginning to tell on the cervix, the abortion may be regarded as inevitable. (2) *Dilatation of the cervix.* This can be felt by the fingers which embrace it in the vaginal roof, and though cases have been noted where the external os has been so expanded that the lower end of the ovum could be felt through it, and yet the pregnancy has gone on to the full time, such a possibility cannot be counted on; and when dilatation is present the abortion should be considered inevitable. (3) *The amount of hæmorrhage* has also to be taken into consideration. There are cases where there has been no appreciable pains, where the cervical canal and its orifices are still contracted, but where the blood-loss calls for active interference. If the amount lost on any one day be not great, yet if it recur time after time, it may be safest for the sake of the mother to further the abortive process instead of allowing her to go on getting her health undermined, to find some months later that she gives birth to a premature or poorly developed child that dies almost as soon as it is born.

PROGNOSIS

However lightly some women may think of a "mishap" as they will call it, the experienced practitioner knows that it is attended with dangers both immediate and remote. Although rarely fatal, deaths have occurred from the severity of the hæmorrhage and from a septicæmia set up by septic abortion. But short of the supreme risk, there is the danger that the hæmorrhage may so reduce the patient's strength that years will pass ere it be regained, and she is rendered liable to crural phlegmasias and pelvic inflammations. There is danger also that the involution of the uterus remain imperfect, so that it is permanently hypertrophied and perhaps displaced. Hence the gynecologist traces back many cases of chronic pelvic distress to the effects of a neglected abortion.

TREATMENT

The treatment of cases of abortion varies acccording as the abortion is—1, Threatened; 2, Progressive; 3, Delayed; 4, Incomplete; 5, Recurrent.

1. THREATENED OR EVITABLE ABORTION.—These are usually cases where there is some degree of hæmorrhage, with uterine contractions in abeyance, and the cervix undilated.

To arrest further mischief, the patient should be kept at complete rest until all hæmorrhage and pain have ceased. The diet should be light and the bowels carefully regulated, and sedatives administered to relieve pain and allay muscular contraction, *e.g.* Tinct. opii ½ to 1 drachm, or an equivalent of morphia by the mouth or hypodermically. A general anæsthetic is sometimes of value until the sedative begins to act. Viburnum prunifolium (ext. liq. ½ to 2 drachms t.d.s.) is frequently serviceable in cases where the discharge continues in small amount, probably due to its sedative action on involuntary muscle. Bromides also may be tried. There are a few cases with marked flaccidity of uterine walls when small doses of ergot (ext. liq. ♏x.), continued for two or three days, check the hæmorrhage. Other styptics, *e.g.* sulphuric acid, etc., may be tried, but are usually worthless. Vaginal plugging may be resorted to for the arrest of the hæmorrhage, but it is of greater service in cases of inevitable abortion.

2. Actual or Inevitable Abortion.—Where the pains are persistent, where the cervix is expanding, or where without notable uterine effort there is free or frequent hæmorrhage, we judge the abortion to be inevitable. An active line of treatment must now be adopted. Three indications have to be fulfilled:—1st, to procure uterine activity; 2nd, to restrain hæmorrhage; 3rd, to secure complete evacuation of the uterus. It hardly needs to be insisted on here that in all the measures we adopt, we must seek to ensure an aseptic delivery with as much care as in the management of a full-time labour. With this view the treatment may be begun with the douching of the vaginal cavity for a quarter of an hour or twenty minutes with an antiseptic lotion of bichloride or biniodide of mercury (1 in 2000-4000) or of carbolic acid (1 in 60-80), or of Condy's fluid, boracic acid, or even vinegar.

1*st. Promotion of Uterine activity.*—Douching has a notable influence in stimulating and strengthening the action of the uterine muscle, especially when the stream of water is of a temperature of from 110° to 120° F. This may be further increased by the administration of ergot in full doses. One or two drachms of the liquid extract may be given, and its effect kept up by repeated half-drachm doses every six or eight hours. Or three to five grains of ergotin may be administered hypodermically, and the effect maintained by the use of ergotin (5 gr.) suppositories. Quinine in two- to six-grain doses every four or six hours may be exhibited with the same good result. The contractions of the uterus are further aided by the means used for fulfilling the next indication, viz.:—

2*nd. Restraint of Hæmorrhage.*—Whilst the uterus is contracting and separating the membranes, especially at the serotinal area, bleeding goes on, and we may think of arresting it, first of all, by packing the vagina with a tampon. We may be called to see a patient without any warning as to the nature of her ailment and have to content ourselves with whatever material in the form of handkerchiefs or pieces of cotton the household may afford. But at least we will do well after douching the vagina to soak the material with which we proceed to plug it in some disinfectant or boil it in a pan to sterilise it. Where we have the opportunity, it is better to use iodoform gauze or some antiseptic cotton pledgets; and wherever we have applied a temporary tampon we ought to return within twelve hours prepared to make a more effective application. Sometimes hydrostatic bags have been used to plug and distend the vagina, or in packing with gauze or cotton through a speculum some of the material may be pushed into the cervix. But the most effective control of the hæmorrhage is attained when the cervical canal is plugged with a tent of tupelo or of prepared sponge. The tent must be carried clear through the cervix so that the distal extremity goes well into the cavity of the uterus whilst the proximal end is just within the os externum. An aseptic sponge-tent of the thickness and length of the middle finger, well planted in a three months' aborting uterus, has the threefold effect of (*a*) absolutely arresting the hæmorrhage; (*b*) stimulating the uterus to contract; and (*c*) dilating the whole canal. If its expansion be hastened by injection of carbolised water into the vagina every half-hour or so, it will so swell up in the course of five or six hours that there is room for the escape of the ovum, which may be driven out under the uterine effort, or extracted, if need be, by the medical attendant.

3rd. *Evacuation of the Uterus.*—Complete evacuation of the uterus *at this stage* is of supreme importance. When the ovum is undergoing extrusion, the canal is more dilatable and the cavity more accessible than it will be later. The patient or attendant must be warned to preserve whatever comes away in such cases, for examination of the products of the abortion usually gives a good idea as to whether the uterus has been fairly cleared. If there be any doubt, the finger should be passed into the cavity of the uterus to explore the walls and corners. If any fragments of membrane are left, and still more if the entire ovum and its investments are still *in utero*, immediate clearance of the cavity should be effected. For this purpose the patient should be anæsthetised. Access to the cavity of the uterus is best obtained by passing a finger or fingers of the right hand through the vagina and cervix whilst the body of the uterus is being pressed down with the left hand applied above the symphysis pubis. The index or medius alone may suffice for the uterine exploration in a two months' abortion. At and after the third month the two fingers must be introduced through the cervix, sometimes with the other fingers and the palm of the hand carried into the vagina. At the fifth month and onwards, the whole hand may have to be passed into the uterus to secure its satisfactory clearance. The fingers must be passed carefully and determinedly round the whole circumference of the ovum so as to detach it and its membranes, especially in the placental area, completely from their uterine detachments. The temptation arises when the practitioner feels that he has got nearly round the ovum to seize it between the fingers and pull on it, in the expectation that in coming away it will bring with it the still unseparated lobules of the placenta. If he take the trouble to examine the mole he has removed, however, he is very likely to find that he has left some placental fragments. He must then introduce his fingers anew for their detection and removal. It is far more satisfactory when the fingers are first introduced to complete the detachment of the ovum from the whole uterine surface, and not to attempt its extraction until the mass lies loose in the cavity. It may now be extracted by laying hold of it between the fingers, or by hooking the two fingers over it and pressing downwards against the front wall of the uterus, whilst the left hand compresses the organ through the abdominal walls. Sometimes the uterus can be freed of its loosened contents by compressing it between the two fingers of the right hand, applied through the posterior fornix vaginæ and the left hand above the pubes; or the loose structures may be withdrawn by means of a pair of polypus forceps or long dressing-forceps; or they may be partly washed out, partly expelled by uterine effort, under the influence of a hot intra-uterine douche. Special curettes and abortion forceps have been contrived, but no instrument can take the place of the sentient finger.

There are patients in whom the abdominal walls are so thick that the hand applied to the hypogastrium fails to press down the uterus within reach of the fingers in the vagina. In such cases it is well to pull down the uterus with a volsella till the external os appears at the vulva. This enables the fingers to thoroughly explore the fundus.

In every instance where fingers have been in the uterus, the cavity should be washed out immediately afterwards with a hot antiseptic douche, a Fritsch or Budin tube being used to allow of the free outflow of the fluid. Its effect is threefold. It washes away any clots or loose fragments of membrane that may have been left *in utero*; it is a prophylactic against septic development; and it stimulates the uterus to energetic contractions. The uterine contractions may be further ensured by a hypodermic injection of ergotin. The cases are very rare where it is necessary to plug the cavity of the uterus; but it is well always to be provided with strips of iodoform gauze against this contingency.

3. Deferred Abortion.—The expression "Missed Abortion" is applied to cases where an ovum has been addled, but not expelled. Not infrequently it is when the gestation-clock strikes the usual full-time hour that the uterus wakens up to expel its long lifeless contents. Occasionally the full term has passed some time before the patient comes to seek an explanation of her disappointment. The diagnosis may be more difficult from the occasional occurrence of a discharge of blood simulating a menstrual period. But a bimanual examination will usually show that the size of the uterus does not correspond with the supposed stage of pregnancy. Where doubt remains, a month may be allowed to elapse. Means should then be taken to evacuate the uterus. The prolonged retention is a continual source of danger, as at any time hæmorrhage may set in, or the uterus may alternately be left in a state of sub-involutionary hypertrophy.

4. Of Incomplete Abortion.—When any part of the ovum has been retained in the uterus, the abortion is said to be incomplete. If there is a history of abortion with the continuance of a sanious discharge or subsequent irregular hæmorrhage with the development of menorrhagia and metrorrhagia, retention of part of the ovum should be suspected. This will usually be found in the serotinal area.

A malignant degeneration of the retained decidua has been described under the term Deciduoma Malignum. The diverse views entertained regarding it will be discussed later.

The treatment consists, in the first place, of making a thorough examination bimanually, and with the sound under the influence of an anæsthetic. If the diagnosis is confirmed, the treatment is curettage. If the cervical orifice is too narrow, it should be dilated with graduated bougies or Ellinger's dilator or a tent. If the practitioner operates towards the end of a menstrual epoch, when the canal is more pervious and its walls relaxed, he will be more successful with the employment of the curette. The expectant method of treatment consisting in the use of hot douches and ergot is likely to be quite unsatisfactory.

5. Of Recurrent Abortion.—A patient who has aborted should be warned against the risk of the recurrence of the accident in the event of a future pregnancy. At all the dates that would correspond to her menstrual epochs, and especially at the date of the occurrence of her abortion, she should be careful to avoid all severe exertion, to regulate properly her food and her evacuation, and to keep absolutely at rest on the faintest indication of any tendency to abortion. Usually when such a patient has reached the mid-term of pregnancy without mischance, she may be encouraged to more freedom, in the expectation that all will go well through the remaining months of her gestation. Where a succession of pregnancies end abortively, and the patient is said to have the abortive habit, we are safe in concluding that she is the subject of some local or general disease. In a very large proportion of cases syphilis is to blame. For such a state of matters abstinence must be enjoined until the poison in the system of both parents is worn out or attenuated. The years of abstinence may be shortened to some months if both the partners are subjected to a systematic mercurial treatment, followed perhaps with iodide of potassium. Where in a patient who has been so treated conception again occurs, it is well to let her take

mercury in tonic dose in the early part of gestation, and from at latest the mid-term onwards to let her take fifteen grains of chlorate of potash three times a day until her child is born. The administration of this salt has also been found serviceable in other cases of abortion and premature death of the fœtus, where there was placental disease of non-syphilitic origin, because of its power of promoting the processes of interchange between maternal and fœtal blood that go on in the placenta. If any morbid local condition exist, such as endometritis, or uterine displacement, appropriate treatment should be carried out.

THERAPEUTIC ABORTION

This term has been fittingly applied by Pinard to the legitimate procuring of abortion by a medical practitioner, with the intention of saving a patient whose health and life are endangered by some of the concomitants of pregnancy. It is a great responsibility that a man takes upon himself when he advises and proceeds to bring about the interruption of a gestation. No wise man will undertake such an operation without the counsel and co-operation of a brother practitioner. The indications for and methods of procedure are described under "Obstetric Operations."

MEDICO-LEGAL ASPECTS OF ABORTION

The illegal procuring of abortion has been practised from the earliest times. It is carried out among the most diverse races in all quarters of the globe, and among all classes in a community. The frequency of criminal abortions in England came prominently to view in June 1898, when Dr. Collins was sentenced to seven years' penal servitude for having procured abortion in a married lady who died of septic peritonitis, and when the jury took occasion "to express their deep concern at and condemnation of the growing tendency on the part of certain classes of the community, as proved by the evidence in this case, to avail themselves of their marital rights, and to try to evade the responsibility of their acts."

In December 1898 three brothers Chrimes were sentenced to penal servitude—the two elder for twelve, and the youngest for seven years—for blackmailing women who had been foolish enough to purchase some of the medicines that are so widely advertised for female "irregularities." They had addressed missives demanding two guineas on pain of arrest to such a number of these deluded women that after the police had got possession of the office hired by the miscreants, they intercepted in the course of two days as many as six hundred letters, each containing two guineas.

The women who subject themselves to this nefarious practice may be unmarried, or widows, or wives living apart from their husbands, who desire to be freed from the fruit of illicit intercourse. Too frequently women living in lawful wedlock are so depraved as to seek the aid of the abortionist when they have conceived in spite of the use of what they had trusted in as preventives. The abortionist may be a degraded, perhaps, like Collins, a dis-registered, medical practitioner. His guilt comes to light when one of his patients becomes the subject of some dangerous or deadly illness, and a consultant is called in to see her. The woman is likely then to confess the origin of her trouble, and the consultant finds the uterus still large and hæmorrhagic. When the patient dies, the hypertrophic uterus will be found showing traces of recent deciduation, and in some cases it is imperfectly evacuated. The genital organs may show traces of damage from the maladroit use of implements. In the case for which Whitmarsh was sentenced in October 1898 for causing the death of a young unmarried woman in whom he had illegally procured abortion, death was due to mercurial poisoning. After using a sound he had injected corrosive sublimate, probably as an antiseptic, but in such strong solution that it had caused sloughing of the canals and allowed of the absorption of the poison into the system. If the medical man who has procured the abortion has alleged the existence of local disease such as perimetric adhesions of a displaced uterus as the excuse for his interference, the post-mortem examination will usually reveal the futility of the allegation. If it is alleged that the patient has been the subject of some general condition calling for interruption of gestation, it will be necessary to adduce evidence other than that of the accused. And in any case it should tell against a medical man suspected of having procured abortion in a case of simple pregnancy that he has had recourse to such a questionable procedure at his own instance and without consultation with another practitioner.

The abortion is sometimes brought on at the instance of the woman herself, sometimes at the suggestion of the father of her child; and the means employed may be self-adopted, or may be prescribed, administered, or applied by another. In some instances women have feigned to have aborted in order to incriminate some one, or to secure compensation for the alleged damage in a railway or other accident. In others, women who mistakenly supposed themselves pregnant have damaged their health by the use of drugs or even produced fatal injuries by the employment of instruments intended to procure abortion.

The means employed to bring about abortion are very various. A long array of drugs has been enumerated that are supposed to have abortifacient properties. Those in popular estimation are ergot, savin, rue, tansy, cotton-root; and aloes, colocynth, and other powerful purgatives. Where drugs fail of their effect, recourse will be had to frequent hot or cold or medicated vaginal douches, or to the introduction of irritant pessaries, or the woman is subjected to severe exertions, riding on horseback, leaping from a height, and such like. More direct measures are employed with greater effect. Tents of tupelo or sea-tangle are introduced into the cavity, which produce their effect by expanding the cervix and stimulating the uterus. Or a sound is passed into the cavity and moved around the walls so as to separate the ovum from its attachments, or to burst the membranes of the ovum and allow of the escape of the liquor amnii. For this last purpose women have been known to make use for themselves of a knitting needle, a crochet needle, or an umbrella rib.

The apparatus of an abortionist may be illustrated by the contents of the bag of a woman Graham who was sentenced in Edinburgh in December 1897 to five years' penal servitude for the illegal procuring of abortion. Besides a pessary or two and some condoms, she had an ergot bottle, a box of aperient pills, a syringe, some tupelo tents, a speculum, a sound, and an old gum-elastic male catheter and stilet. No woman who was not following the felonious practice of an abortionist could be possessed of such a set of implements, and every owner of such a bag would suffer no injustice if she were sent for five years to a felon's cell.

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Abscess. *See* SUPPURATION.

Absinthism.—The use of absinthe as a beverage is especially prevalent in France. The practice of absinthe drinking is said to have been introduced into the country after the Algerian War (1844-47) by the soldiers, who had acquired the habit of using it as a febrifuge during the campaign. The use of it rapidly increased, and with such disastrous results that it has been described by French physicians as constituting a graver danger to public health than alcohol itself.

Composition.—Absinthe is a liqueur, of an emerald-green colour, containing a high percentage of alcohol, and various essences obtained by macerating in alcohol parts of several plants, especially species of wormwood (*Artemisia*), and star-anise (*Illicium anisatum*); also sweet flag root (*Acorus calamus*), and angelica root (*Archangelica officinalis*).

Symptoms.—Absinthism differs in many respects from alcoholism. In absinthism terrifying dreams and hallucinations, intellectual enfeeblement, stupor, may come on rapidly and in the entire absence of muscular tremor. If any tremor is present, it is usually confined to the upper extremities.

Absinthe tipplers suffer from restlessness at night, dreams, anorexia, nausea, and vomiting, mental dulness, giddiness, and convulsions. Delirium or mania may occur, and eventually the patient may die in a condition of idiocy.

The epileptiform convulsions form a very characteristic feature in absinthism, and are readily produced in animals.

Mode of Action.—The phenomena of absinthism are due to the stimulation of the cerebro-spinal nerve-centres, and especially, as Magnan long ago proved by experiments on animals, of the bulbo-cervical region of the cord. The delirium and hallucinatory disturbances are due to a stimulation of the cerebral-cortex, similar to that produced by haschish (*Cannabis indica*), belladonna, and hyoscyamus.

Although some French authors have maintained that the toxic effects of absinthe are simply those of the alcohol it contains, there seems to be no doubt that the nocuousness of absinthe far exceeds that of simple spirits. The special effects of absinthe appear to be due to the essence of absinthe, and perhaps to the essence of anise, which it contains. Much of the absinthe sold is adulterated. Copper salts even have been added to produce the green colour. According to Bernard Roux, wormwood (*Artemisia absinthium*) contains two active principles: a volatile oil, the essence of absinthe, and absinthin. The former alone is capable of producing an intoxication characterised by epileptiform convulsions.

Treatment.—(*See* "Alcoholism").

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Acanthosis Nigricans. *See* SKIN.

Acclimatisation. *See* CLIMATE.

Accommodation.

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THE refractive power of the dioptric media of the eye is not fixed; by an effort it can be increased, and when the effort is relaxed it returns to its previous state. This increase in the refractive power is termed accommodation. An individual with normal refraction is one who can focus parallel rays on his retina when the eye is at rest; he is thus able, without any effort, to see distinctly objects in the distance, since rays coming from the distance are practically parallel. But in order to focus on his retina the diverging rays from objects near at hand, the refractive power of the eye must be increased. When, therefore, he wishes to see near objects distinctly an effort of accommodation has to be made.

In order to obtain a clear idea of the opinions held on accommodation at the present time, it will be well to note first some of the chief anatomical relations of the parts concerned.

The ciliary muscle lies in the outer part of the ciliary body, next to the sclerotic. Its fibres may be roughly divided into two parts—(*a*) an external set, whose course is longitudinal, attached in front to the fore part of the sclerotic between the canal of Schlemm and the anterior chamber, and behind to the choroid opposite the ciliary processes and partly farther back towards the ora serrata; (*b*) an internal bundle, whose course is circular, running close to the insertion of the iris. In addition to these two sets, radial fibres exist which, starting from the sclerotic in company with the external longitudinal fibres, pass inwards to join the circular bundle.

The crystalline lens, enclosed in its highly elastic capsule, consists of a central, more resistant part—the nucleus—which does not alter its shape, and a peripheral, semi-gelatinous zone—the cortex—which by pressure or traction can readily be made to change its form. The lens rests posteriorly in the hollow in the fore part of the vitreous body, to which its capsule is closely adherent; it is also kept in position by means of the suspensory ligament.

The suspensory ligament, consisting of a series of fine fibres radially disposed, is attached in front to the capsule of the lens, chiefly to the anterior surface near the margin, partly also to the margin and posterior surface. Behind it is adherent to the pars ciliaris retinæ from the ora serrata to the most prominent parts of the ciliary processes.

We are now in a position to consider the more important views put forward to explain the act of accommodation.

Some of the older views need only be referred to briefly. At different times it has been thought that accommodation was due to—

(1) *Elongation of the Globe* by combined compression and traction of the external and internal muscles of the eye respectively. Young proved that no elongation took place during accommodation by experiments on his own eye.

(2) *Alteration in curvature of the Cornea.*—Young found that with his eye under water, thus practically nullifying the refractive power of the cornea, his accommodation remained unimpaired.

(3) *Contraction of the Pupil.*—Von Graefe's case of absence of iris due to traumatism, in which there was full power of accommodation, shows this view to be incorrect.

(4) *Alteration in position of the Lens.*—The lens moves forwards about ·5 mm.

during accommodation; but if it came forward so as to touch the cornea, the increase in the refractive power gained would be much less than that in accommodation.

(5) *Alteration in the curvature of the Lens.*—If a bright light be held in front of an eye in a dark room with proper precautions, three reflected images are visible; one image, distinct and upright, reflected from the anterior surface of the cornea; another, much smaller and inverted, reflected from the posterior surface of the lens; and a third image, larger and upright, but less easily seen, reflected from the anterior surface of the lens. When the eye accommodates, the image from the cornea does not change; that from the posterior surface of the lens is altered very slightly, or not at all, while that from the anterior surface of the lens becomes distinctly smaller. This observation proves that during accommodation a change takes place in the lens of such a nature that its anterior surface becomes more convex; it further proves that there is no change in the curvature of the cornea, nor to any marked extent in that of the posterior surface of the lens. An increase in the convexity of the anterior surface of the lens necessarily causes an increase in the refractive power of the eye. The question arises, can the whole of the accommodative power be accounted for in this way? Knapp observed in four individuals the difference between the curvature of the anterior surface of the lens when at rest and when in a state of extreme accommodation, and calculated the range of accommodation that might be expected. He then tested the actual range of accommodation and found that it corresponded fairly closely with the range calculated. We may take it, then, as proved that the change in curvature of the anterior surface of the lens gives a sufficient variation in refractive power to account for the whole range of accommodation. We must now consider how this change is brought about.

It is at present generally accepted that the change in shape of the lens is dependent upon the contraction of the ciliary muscle, but the exact manner in which this change is effected is still a subject of controversy. The older views, that the iris or ciliary body pressed on the lens and so caused the change in shape, are untenable, since accommodation takes place in the absence of the iris, and the ciliary processes do not touch the lens.

Before discussing the two theories put forward to explain the mechanism of accommodation, which have received most support, viz. those of Helmholtz and Tscherning, it will be well to tabulate the more important observations that have been made.

(*a*) *Changes in the Lens.*—Its *anterior* surface moves forwards about ·5 mm. during accommodation, and at the same time becomes more convex, its radius of curvature changing from about 10 mm. to 6 mm. Tscherning, from very careful observations of images reflected from different parts of the anterior lens surface and from entoptic phenomena, came to the conclusion that the increase in convexity was not uniform, but more in the centre than at the periphery. In many of his observations he followed the experiments of Thomas Young. He estimated that at the centre the radius of curvature changed from 10 mm. to 5 or 6 mm., at 1·4 mm. from the centre the radius was only 8 mm., while at the periphery the curvature was actually diminished. These observations have been confirmed by Crzellitzer. The radius of curvature of the *posterior* surface changes very slightly, possibly from 6 mm. to 5 mm.; the position of this surface is probably not altered. Hess and Heine have observed that the lens becomes more *movable on accommodation*, and that it is displaced slightly in a direction determined by gravity; this mobility was more marked in an eye under the influence of eserin, which causes spasm of accommodation. In cases of rupture of the suspensory ligament the lens becomes more globular and the refraction of the eye more myopic.

(*b*) *Changes in the Choroid.*—Hensen and Völkers noticed a movement forwards of the choroid on accommodation, greatest in the region of the equator.

(*c*) *Changes in the Iris.*—The pupil contracts with accommodation and the iris is pushed forwards by the bulging lens near its pupillary margin. Cramer and Tscherning describe a circular groove in the iris during accommodation near its attachment, with the concavity directed backwards.

(6) *Intraocular Tension.*—Manometrical observations, taken simultaneously in the anterior chamber and in the vitreous body, show no difference of pressure at these situations, during accommodation. But in cases of keratocele, recession of the bulging membrane of Descemet has been observed by some during accommodation, which has been taken to indicate a relative fall of tension in the anterior chamber.

(7) Iwanoff describes a great development of the circular fibres of the ciliary muscle

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in hypermetropes, who use their accommodation a great deal, and a very slight development in myopes, who use their accommodation very little. This change, however, is said not to be constant, and in nearly all mammals, whose mechanism of accommodation is probably the same as in man, the circular fibres are either absent or nearly so.

Of the two main theories of accommodation, that of Helmholtz is the older and the one generally accepted.

Theory of Helmholtz.—According to this, when the eye is at rest, the suspensory ligament is in a state of tension, and exercises traction on the anterior capsule of the lens, thereby causing this body to become flattened anteriorly. The ciliary muscle has its more fixed attachment in front, and therefore, by the contraction of the longitudinal fibres, it draws forward the choroid, and with this the suspensory ligament. The ligament is now relaxed, and the elastic lens, being released from the traction, becomes more convex anteriorly, and with this change its refractive power is increased. When the ciliary muscle relaxes, the suspensory ligament once more becomes taut, the anterior surface of the lens consequently becomes flattened, and the refractive power is decreased.

Theory of Tscherning.—This is in certain respects diametrically opposed to the theory of Helmholtz. Tscherning bases his view on the observations mentioned above—that only the central part of the anterior surface of the lens is increased in convexity during accommodation, while the peripheral zone is flattened, a change which he maintains could not be brought about by relaxation of the suspensory ligament. Tscherning considers that by the contraction of certain deep fibres of the ciliary muscle which he describes, the ciliary processes are drawn outwards and backwards, and by this movement the suspensory ligament is rendered more tense. At the same time the longitudinal fibres draw the choroid forwards, and thereby give support to the vitreous body and indirectly to the lens itself. As a result of the tension of the suspensory ligament, the lens, supported from behind, is moulded anteriorly in a twofold manner, the peripheral zone is flattened, while the central portion is rendered more convex.

Thus, according to Helmholtz the suspensory ligament is slackened during accommodation, whereas according to Tscherning it is tightened.

Among the various points in favour of the older view may be mentioned — the globular shape taken by the lens on rupture of the suspensory ligament, and the high degree of myopia which is developed; also the greater mobility and sinking of the lens on accommodation, an observation most difficult to explain by Tscherning's theory. It must be allowed that further knowledge is necessary before the matter can finally be decided; meanwhile the balance of opinion remains in favour of the classical view of Helmholtz.

NERVOUS MECHANISM OF ACCOMMODATION.—The ciliary muscle is governed by a centre, which in dogs is situated at the foremost part of the nucleus of the third nerve in the floor of the central canal opposite the junction of the aqueduct and the third ventricle. Stimulation of the centre, or the third nerve, or short ciliary nerves, leads to contraction of the ciliary muscle and accommodation for near objects, thereby indicating the path taken by the ciliary fibres. Whether relaxation of accommodation is also under special nervous influence has not been definitely decided. Jessop observed recession of the anterior surface of the lens on stimulating the long ciliary nerves, after the ciliary muscle had been partly contracted with pilocarpin. Morat and Doyon record enlargement of the reflected image from the anterior surface of the lens in dogs and cats which were under the influence of morphia or pilocarpin. On the other hand, Langley and Anderson failed to observe either backward movement of the lens or enlargement of the lens images. The actual time taken in relaxation of accommodation is much shorter than that taken in accommodation itself, a fact which is not in favour of the view that the former is simply passive.

UNEQUAL ACCOMMODATION.—By this is meant an unequal degree of accommodation in the two eyes at the same time—a condition which might be expected to occur in cases of anisometropia (unequal refraction in the two eyes) in order that the focus in the two eyes might be equalised.

From the observations of Hering and Donders it would appear that unequal accommodation does not occur; the later experiments of Fick tend to a contrary opinion, but, again, Hess's very careful experiments support the older view. The latter experimented with very fine threads, which were only visible when they were situated in the focal plane of the eye; he arranged them in the stereoscope, so that only one thread was seen by each eye. One thread being kept stationary, he found that in cases of anisometropia a deviation of the other thread, corresponding with a lens of ·5D, from the focal plane of the eye observing it, caused the thread to become indistinct, or even invisible—showing that no variation in the accommodation of the two eyes could be made.

UNEQUAL CONTRACTION OF DIFFERENT PARTS OF THE CILIARY MUSCLE.—Some have thought that such a condition might occur on the grounds that (*a*) an increase in the amount of astigmatism has been noted in some cases after instillation of atropin; (*b*) the axis of astigmatism has been noticed to vary at different times. On the other hand, Hess, in his experiments, failed to find any evidence in favour of unequal contraction sufficient to overcome even a slight degree of astigmatism. He used two extremely fine threads, one at right angles to the other, and placed them in front of an astigmatic eye, so that one thread was in the focal plane of each astigmatic meridian. In that position both were seen, but directly one of the threads was moved forwards or backwards, there was at once blurring of the thread, and by no effort of accommodation could both threads be focussed again. He also found that astigmatic individuals do not accommodate for one or other of their linear foci, but for an intermediate, approximately circular focus between the two linear foci. That the most distinct image is obtained in this way he showed by rendering a photographic apparatus astigmatic; the clearest picture was obtained when the apparatus was adjusted at the circular focus intermediate between the two linear foci.

AMPLITUDE OF ACCOMMODATION.—This is the total accommodation of which an eye is capable; it is therefore equal to the difference between the refractive power of the eye at rest and when accommodated to its fullest extent, that is to say, when accommodated for its near point. Let us consider the measurement of the amplitude of accommodation in emmetropia, hypermetropia, and myopia.

In emmetropia, parallel rays are focussed on the retina by the refractive power of the eye at rest, and therefore the amplitude of accommodation is equal to the accommodative effort made in focussing objects at the near point. Suppose an emmetropic eye has its near point at 20 cm., and a lens of 20 cm. focal length be placed immediately in front of it; the lens will cause the transmitted rays starting from the near point 20 cm. distant to be parallel, and the eye can focus them without any effort of accommodation. Thus it is seen that a lens of 20 cm. focal length has done the work equal in amount to the fullest effort of accommodation. The amplitude of accommodation in this case is therefore equal to a lens of 20 cm. focal length, which is a lens of 5D.

In the case of hypermetropes, the hypermetropia has to be overcome by the accommodation in order to focus distant objects. The amplitude of accommodation will therefore be the sum of the hypermetropia, and that increase in the refractive power of the eye required in accommodating from distant objects to those situated at the near point. Let us suppose, for example, a hypermetropic eye of 2D has its near point at 25 cm. A hypermetropic eye of 2D is one which has to accommodate 2D in order to focus parallel rays. As explained above, the effort of accommodation required to focus rays diverging from a point 25 cm. over the amount required to focus parallel rays will be equal to a lens of 25 cm. focal length; for the lens of 25 cm. focal length placed in front of the eye will give the transmitted rays from a point 25 cm. distant a parallel direction. Now a lens of 25 cm. focal length is a lens of 4D, therefore the total accommodative effort exerted by the hypermetropic eye of 2D in focussing for its near point, 25 cm. distant, is 2D + 4D = 6D, which is its amplitude of accommodation.

In myopia the amplitude of accommodation is the increase in the refraction of the eye when focussing from the far point to the near point. Let us take as example a myope whose far point is at 25 cm. and his near point at 10 cm. It is obvious that the increase in refraction is exactly the same for a myope focussing from a point 25 cm. to a point 10 cm. distant as for an emmetrope. Now the accommodation required by an emmetrope to focus objects at 25 cm. is equal to a lens of 25 cm. focal length, *i.e.* a lens of 4D, and the amount required to focus objects at 10 cm. is equal to a lens of 10 cm. focal length, *i.e.* a lens of 10D. The difference (10D − 4D = 6D) represents the increase in the refraction required in focussing from 25 cm. to 10 cm.; and therefore the amplitude of accommodation of the myope, whose far point is at 25 cm. and near point at 10 cm., is equal to 6D.

The amplitude of accommodation varies with age, and is practically unaffected by differences of refraction; thus an individual of twenty years of age will have the same amplitude of accommodation whether he be emmetropic, hypermetropic, or myopic.

PRESBYOPIA.—The nucleus of the lens is formed by the gradual sclerosis of the central parts, and as age advances the nucleus gradually increases in size. In children no distinct nucleus exists, though the centre of the lens is more resistant than the periphery. At twenty-five years a distinct though small nucleus is found, while in advanced age very little cortex remains, practically the whole of the lens having become sclerosed. The amplitude of accommodation varies with the amount of soft cortex, and thus as age advances the amplitude gradually diminishes, and with this the near point recedes. Donders discovered this fact, and determined the average amplitude of accommodation at different ages. He found that at ten years there is an amplitude of 14D, at twenty years 10D, at 30 years 7D, at 40 years 4·5D. After forty years of age the accommodation loses on the average 1D every five years, and after sixty there is very little power of accommodation left.

In order to do sustained work with comfort at 30 cm., the near point should not be farther away than 22 cm., as it is well to have a little accommodation in reserve. Now an emmetrope of forty years of age has his near point at 22 cm.; as his age increases the near point recedes and close work becomes difficult, especially reading fine print or threading needles by gaslight. The work is first held farther off, later on close work becomes impossible without the help of glasses. The actual changes which have led to this result began in childhood, but it is only when the near point has receded beyond the position at which close work is habitually performed that the individual is inconvenienced, and it is then that *presbyopia* is said to have commenced.

As above stated, for sustained close work the near point should not be beyond 22 cm.; for an emmetrope to see at this distance an accommodation of 4·5D is required. At forty-five years his power of accommodation is reduced to 3·5D, and he will therefore need a convex lens of 1D. At fifty, having only 2·5D of accommodation, a lens of 2D is required; at fifty-five a lens of 3D; at sixty a lens of 4D. After the age of sixty the eye becomes slightly hypermetropic, and therefore rather a stronger glass may be required, even though the amplitude of accommodation, being practically nil, can no longer change.

It is clear that in hypermetropia, presbyopia will appear earlier, and in myopia later than in emmetropia; indeed a myope of 4·5D does not need to wear glasses for close work. If, however, the full correction of the H. or M. be worn, presbyopia is found to set in at the usual age. In astigmatism the correcting cylinder should be added, and in hypermetropia the corresponding power should be added to the presbyopic glass; in myopia this should be subtracted. In ordering glasses for close work each case must be tried separately. Donders' table of the amplitude of accommodation at different ages gives an indication, and a very accurate one, but individual variations exist. Thus hypermetropes, with their well-exercised power of accommodation, often prefer a much lower lens, and an individual "older than his years" prefers a stronger lens than one would expect.

Accommodation and Convergence.—With each degree of convergence of the visual lines a certain effort of accommodation is associated; but the connection is somewhat elastic, for without altering the angle of convergence the accommodation may to a certain extent be increased or diminished. This may be shown by looking at an object at about 2 m. distant, and holding first weak convex and then weak concave glasses before the eyes, when the object can still be seen distinctly. The strongest convex and the strongest concave lens which can thus be used represent the measure of the amplitude of accommodation for this point. For each degree of convergence there is a *relative amplitude of accommodation.* The part of this amplitude of accommodation in use when looking at the object (without any glass), and represented by the convex lens, is called the *negative* part; while that represented by the concave lens is called the *positive* part; it has not been brought into play, but is, as it were, held in reserve. It is essential for sustained accommodation at any angle of convergence that the positive part of the relative amplitude of accommodation be considerable. When the correcting glasses for an error of refraction are worn, it is obvious that the previous relation between convergence and accommodation is upset; this probably accounts for much of the trouble felt in wearing correcting glasses for the first time. That the connection between convergence and accommodation is not rigidly fixed, is also shown by the fact that if weak prisms with their bases placed inwards or outwards be held up before the eyes, when looking at an object, distinct vision can still be maintained. The prisms cause the visual lines to be more convergent or less convergent, according as their bases are placed outwards or inwards, without altering the refraction. This shows that convergence of the visual lines may vary while the accommodation remains constant.

Spasm of Accommodation.—In speaking of spasm of accommodation, three separate conditions must be distinguished—(*a*) tone of the ciliary muscle; (*b*) habit spasm; (*c*) true spasm.

(*a*) *Tone of the Ciliary Muscle.*—In young people the instillation of atropin, besides causing paralysis of accommodation, usually brings about a slight change in refraction; thus an emmetrope becomes slightly hypermetropic, hypermetropes and myopes become slightly more hypermetropic and less myopic respectively. This is due to the ciliary muscle having lost a certain contraction which remained unrelaxed even with the eye at rest, and which is spoken of as tone of the ciliary muscle. In children this usually amounts to about ·75D; it diminishes with age, and is rarely found after thirty-five years, and, indeed, in some much younger people it may not be appreciable.

(*b*) *Habit Spasm.*—In hypermetropes who have not worn correcting glasses, the habit of overcoming their hypermetropia by accommodation is often so pronounced, that when the correcting glasses are first worn they cannot relax their accommodation. This condition may be termed *habit spasm.* As a rule it is only compensatory, and is not in excess of the hypermetropia; and, moreover, in the dark room the spasm relaxes and a just estimate of the hypermetropia may be made by retinoscopy or direct estimation; but directly the individual makes the effort of looking at a distant object through the correcting lenses the habit spasm is set up. If the patient perseveres in wearing the glasses constantly, and especially if helped by instillation of atropin and cessation of all close work for a time, the habit spasm soon subsides.

In myopia, the constant habit of holding objects very close to the eyes sets up an excessive contraction of the ciliary muscle; so that a stronger lens than that representing the myopia is selected. This tendency to overcorrection must constantly be kept in mind when ordering glasses for myopes. The habit spasm here also is relaxed when the eyes are at rest in the dark room.

When possible it is advisable to paralyse the accommodation with atropin or homotropin in all cases of habit spasm before ordering glasses; it ensures more accurate results, and the patients become more quickly accustomed to their glasses.

(*c*) *True Spasm.*—No sharp line can be drawn between this condition and that termed habit spasm. Clinically, however, a distinct class of cases is met with, though rarely, in which the spasm of accommodation is excessive in amount; so that emmetropes and those with low degrees of hypermetropia appear to be myopic, and myopes much more myopic than they really are. They suffer from distressing asthenopic symptoms, the spasm does not relax completely when at rest in the dark room, and the condition is often found to recur even after paralysing with atropin. The subjects of it are frequently of a neurotic disposition.

The *cause* usually assignable is overwork, associated with some refractive error, and in one recorded case there was a slight injury of the cornea. It may be artificially produced by the instillation of eserin.

Treatment.—Correction of the refractive error, together with a course of instillation of atropin, and cessation of all close work, should be advised, and the general health carefully attended to.

Paralysis of Accommodation.—This may be partial or complete; it may affect one or both eyes; it may occur alone, or when associated with paralysis of the sphincter iridis, the condition is known as *ophthalmoplegia interna.* Or, again, it may be associated with paralysis of one or more of the external ocular muscles, usually those supplied by the third nerve.

The symptoms are similar to those described previously in presbyopia, and the condition may be diagnosed by finding that the amplitude of accommodation does not correspond with the age. In myopia the condition gives rise as a rule to very little trouble, and is often overlooked.

Causes.—The three most common causes are—atropin, diphtheria, and syphilis. It may be due to the instillation of atropin, or to one of the other mydriatics, or to large doses taken internally. It may also be due to ptomaine poisoning. In diphtheria it is seldom associated with paralysis of the sphincter iridis.

Syphilis may affect the third nerve anywhere in its course from the centre to the periphery. Other causes may be mentioned—affections of the third nerve apart from syphilis, influenza, exposure to cold, contusion of the eye, diabetes, and degenerative diseases of the central nervous system, especially tabes.

Prognosis.—This must be guarded, as the result is very variable, except in the case of diphtheria, where the prognosis is always favourable.

Treatment.—The cause should if possible be treated; general tonics may be given, and if the condition persists, the patient may be helped by correcting glasses for close work.

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Acetabulum. *See* HIP-JOINT.

Acetonæmia and Acetonuria. *See* DIABETES, URINE.

Achondroplasia.—*Definition.*—Achondroplasia is a fœtal disease which causes a defective growth of certain of the bones *in utero*, and leads to congenital dwarfing of the extremities and other deformities which persist through life.

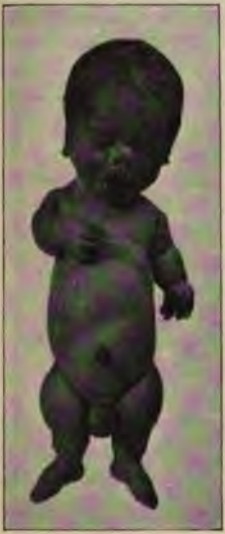
Nomenclature and History.—The condition used to be regarded as a sort of fœtal rickets, until it was pointed out by Depaul and others that the bone lesion was not at all the same as in that disease. It was afterwards thought to be a form of fœtal cretinism (Virchow, Eberth), but this also has been disproved. It is now believed to be an entirely different disease, and a number of names have been applied to it which indicate its effect on the growth of the bones. Achondroplasia (Parrot) is that which has been most used in this country and in France, while in Germany it is more often referred to as Chondrodystrophia Fœtalis (Kaufmann).

As Parrot has pointed out, achondroplasic dwarfs are found in many of the sculptures of the ancient Egyptians; and several of the old masters have introduced figures of this type into their pictures.

Etiology.—The causation is still quite obscure; neither syphilis, alcoholism, nor malnutrition of the parents has anything to do with it. Several writers (Porak, Boeckh) have recorded instances of its being hereditary. It certainly also seems to have a tendency to occur in the same families as cases of sporadic cretinism, infantilism, and ordinary dwarfing. Hence some have attributed it to a morbid action of the thyroid (Hertoghe), but this has not been proved.

Among certain of the lower animals well-marked instances of the condition occasionally occur. It is also interesting to find that there are distinct breeds of dogs (bassets or dachshunds), cattle (natos), sheep, and goats which habitually present most of the peculiar deformities characteristic of achondroplasia.

Time of Origin and Course.—The disease sets in during early fœtal life, and is said to run its whole course between the third and sixth months. At birth, accordingly, we only find the results of past disease, and not an advancing process, as in congenital syphilis or rickets. Probably it is only in the slighter cases that the infant is capable of surviving birth. Hydramnios is very often present, and the labour is generally premature. Abnormal presentations are common, and prolapse of the cord has often been noted. Even if the infant is born alive he is generally so weakly that he dies within a few days. Should the child, however, survive early infancy, the subsequent development of his muscular, cutaneous, and reproductive systems is normal, and his intellect is unaffected. The chances of life in these cases are probably as good as those of an ordinary individual, except in the case of women who become pregnant.



Pathology.—The main abnormality consists in an interference with the growth of certain bones. This is found to be due to the endochondral ossification, by which they normally grow in length, becoming more or less completely arrested at an early period of intra-uterine life. Consequently, the cartilage cells at the epiphysial lines show none of the arrangement into parallel rows which is the preliminary to normal bone-formation.

In the new-born infant this defect is found to be entirely confined to the bones which grow in cartilage during early fœtal life, while those which are formed in membrane are unaffected; and those cartilage-formed bones in which ossification does not begin before birth, or not until late in intra-uterine life, are not involved at all, or only to a slight degree. Thus we find that the clavicle and the cranial vault, also the sternum, costal cartilages, patellæ, carpus, and tarsus, are of normal size, while the long bones of the extremities, the ribs, the innominate bones, and the posterior part of the base of the skull are more or less stunted in growth. In many cases the basis cranii is not only shortened, but its three osseous nuclei are prematurely united. The thyroid gland is unaffected.

Clinical Features.—The deformity which results from the above-mentioned distribution of the bone-lesion is very characteristic. The head is of fully normal size, the cranium being high and bulging in front and at the sides. There is usually an abrupt depression at the root of the nose, and the whole nose and upper jaw may be depressed. The tongue often protrudes. The trunk is of normal length, but narrow from the shortness of the ribs and from the pelvic deformity. The arms and legs are little more than half the normal length, their bones being thick, as well as short, with very large epiphyses, and the curves which they present being exaggerations of those normally present. The limbs are often encircled by deep sulci, as if the skin were redundant. When the palm is flat the fingers do not lie parallel as in a normal hand, but diverge somewhat, two usually turning towards the radial and two towards the ulnar side. There is occasionally some degree of hydrocephalus, and sometimes considerable swelling of the subcutaneous tissues.

A number of cases of short-limbed new-born children have been published which differ from the above description in the following respects. Some of the long bones, especially those of the legs, present acute curvature of their shafts; the epiphyses are not greatly enlarged; the hands do not show any abnormality; and there is usually, if not always, extensive craniotabes. Although these cases have sometimes been described as achondroplasia by good authorities, it seems almost certain that they are examples of a different disease—possibly of real intra-uterine rickets.

When the child grows up, the disproportion between his body and limbs becomes even more noticeable. The head remains relatively large; the nose generally presents a depression at its root, and the lower jaw is apt to be underhung. The tendency for the tongue to be protruded does not, however, persist. When the child learns to walk, the gait is peculiarly waddling, and a deep lumbar lordosis develops. When the arms are by the side, the finger-tips reach to a little below the iliac crests in women, and to about the level of the trochanter in men. The whole lower limb is about the length of a normal thigh. The epiphysial ends of the long bones expand very abruptly. Adults usually measure from 3½ feet or less to a little over 4 feet. Should a dwarf of this type become pregnant, the extreme narrowness of the pelvis generally necessitates operative interference. Typical photographs of adults have been published by Boeckh, Herman, Porak, J. Thomson, and others.

Diagnosis.—At birth the deformity already described forms a picture which is easily recognised. The appearance of these infants has often been termed "cretinoid," and aptly enough, because they are certainly like cretins in several particulars; but it is to be observed that the likeness is to adult cretins, and not to cretin babies. In infancy even severe cases of sporadic cretinism do not present much disproportion of the limbs and trunk, nor yet the typical physiognomy of the disease, except to a very slight degree.

The achondroplasic *adult* differs distinctly from the rickety dwarf in having all his limbs equally shortened, and in the shortening of his hands and the peculiar divergence of his fingers. He also presents a striking absence of rickety distortion in the spine, thorax, and head. On inquiry it will usually be found that the achondroplasic cut his teeth early, and was not late in learning to walk, while rickety dwarfs have almost always been very backward in both these particulars.

From cretins, adult achondroplasics may be readily distinguished by the normal temperature and texture of their skin, their muscular and intellectual vigour, and the natural development of their sexual organs.

Treatment is out of the question. The administration of thyroid substance has no effect on the retarded growth.

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Acids and Acidity. *See* DIGESTION, BLOOD, URINE.

Acids, Poisoning by. *See* TOXICOLOGY.

Acne.

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UNDER the generic name *Acne* the older French school united nearly every pathological form which was supposed to be a functional or structural disorder of the sebaceous system. Modern investigations have tended more and more to break up such a group, so that the term "acne" is now generally reserved for a limited number of affections. Kaposi, however, includes an A. disseminata or vulgaris, varioliformis, cachecticorum, artificialis (from local irritants, such as tar); acne from the ingestion of iodides and bromides (iododerma and bromoderma pustulo-tuberosum, Unna); acne rosacea, and sycosis (A. mentagra). Unna adopts the old narrower definition of acne, which represents a single variety of folliculitis, and expressly excludes folliculitides of other origin. We shall consider here only the common acne, A. varioliformis, and rosacea.

ACNE VULGARIS (FUCHS), ACNE DISSEMINATA (KAPOSI).—*Acne vulgaris* is an affection chiefly of certain definite regions of the skin, viz. the face (forehead, nose, cheeks), the external ears, neck, upper part of the trunk on both aspects, and, more rarely, the whole back and even limbs, occurring during the period of puberty and usually tending to disappear with complete development, but sometimes persisting much later. It is characterised by the presence on an oily, discoloured, thick-looking skin of a polymorphic eruption, presented all together or in successive evolution and in varying proportions; first, black-headed plugs, called *comedones* (A. punctata), in the mouths of the follicles, which exceptionally may go on to form sebaceous cysts, from a pea to a nut in size; secondly, a greater or less abundance of painful, conical or hemispherical, inflammatory papules and superficial or deeper pustules centred by comedones (A. papulosa et pustulosa), from a pin-head to a pea in size, and even small abscesses; thirdly, red, hard, painful nodosities (A. indurata, A. tuberosa), which are true granulomata, according to Unna. These inflammatory elements leave macules and flattened scars, and sometimes a curious deformity by confluence of diseased follicles, known as double or treble comedones. By pressure the contents of the follicles and pustules can be extruded. This affection is said to be most common in dark males. All degrees of severity are met with. Sometimes the comedones and greasy skin are the prominent feature, but, on the other hand, most severe cases may be met with in which the back especially is thickly covered with small phlegmons and abscesses.

Acne is often associated with a defective general nutrition and sluggish circulation, chronic dyspepsia, constipation, anæmia, and menstrual disturbances in the immature subjects it affects, and these conditions, as well as irregularities of diet, self-abuse, or, on the other hand, chastity, have been assigned by various authors as predisposing or exciting or intensifying factors. Kaposi states the old view, that the affection is an anomaly of secretion and excretion of the sebaceous glands, and that the inflammation is provoked in the tissues surrounding the sebaceous glands and hair-follicles with their common excretory duct by the retained secretion, mechanically or by chemical decomposition. It is obvious that the affection occurs at a time when the functions of sebaceous secretion and hair-growth are active, but whether it should be regarded as a local developmental aberration solely, or intensified or excited by general states, is in dispute.

The elaborate investigations of recent years have, however, started other views which are of interest. *Sabouraud* holds that the foundation of the affection is an oily seborrhœa, due to a micro-bacillus, which is associated with the formation of little cocoons in the mouths of the pilo-sebaceous follicles. In support of this view we may note the plugging of the follicles, apart from the formation of comedones and the frequent presence of seborrhœic catarrh of the scalp. Amongst the thousands of cocoons, some enlarge and become monstrous and degenerated (comedones). The latter contain within the much-thickened envelope of corneous epithelium colossal pure colonies of a micro-bacillus, and 2-4-6-8 atrophied, recurved, dead, downy hairs, representing the frustrated efforts at a growth of hair. A comedo is also undergoing degeneration and destruction, and its summit and exterior envelopes are therefore always sown with various secondary infections (one can meet with half a score), which give the characters to the various manifestations of polymorphous acne. Two of these secondary infections are constant, viz. Unna's bottle-bacillus, discovered by Malassez in 1875, which appears to be without pathogenic significance, and a white coccus, different from the white staphylococcus, which apparently causes the intradermic inflammatory nodules and abscesses. In some cases, according to Sabouraud, furuncles form due to the golden staphylococcus lurking in the outer envelopes.

Unna's view is somewhat different. Associated with a frequent activity of the coil glands (hyperidrosis oleosa) there is a hyperkeratosis with thickening of the horny and granular layers, which extends into the follicle mouths and leads to the formation of the horny cysts, called comedones, in the dilated mouths of the common opening of the sebaceous duct with its appended lanugo-hair. The sebum is dammed back and may sometimes, with distorted lanugo-hairs, be enclosed in the cyst, or the latter is generally incomplete at its inner end and terminates in sebum. The black head is not due to dirt but to a diffuse pigment. Almost every comedo contains a swarm of micro-organisms, often in pure culture, especially the kerato-philic oat-shaped bottle-bacillus and the diplococcus of seborrhœic eczema, both chiefly localised in the head and mantle of the comedo, also a special mucus-producing bacillus,[1] which from its constancy and position in the lower part of the comedo is probably the actual cause of comedo-formation and of suppuration, which is *not* due to pyococci. He says there is no tendency to furunculosis. Unna dwells on the presence of a chronic progressive hyperplastic inflammation of the acne-skin (A. hypertrophica), most developed round the follicle, which causes a loss of elasticity and hence a plugging of the follicles with retained sebum. This change is most developed round the follicles and produces, in some cases, the nodules known as acne indurata, which, as Sabouraud also remarks, abound in interesting types of cells. It does not lead to rhinophyma. Lomry concludes that the Staphylococcus albus is constantly present in the comedones and pustules, as in the skin which is not the seat of acne, and it only becomes pathogenic in the presence of a peculiarly favourable soil.

The combination of characters here set forth (morphological and regional), especially the presence of comedones, makes the *diagnosis* easy as a rule, and enables a distinction to be made from acne varioliformis, bromide and iodide acne, and the generalised or specially distributed acneiform syphilides and "tuberculides." A clear conception of certain points will assist, viz. (1) the distinction between the true comedo and the many states of plugging of the follicles with dark-headed collections of sebum or epithelium, accompanied by more or less congestion or inflammation; (2) the differences between the acne or acneiform papulo-pustule, and sycosis and impetigo pustules.

ACNE VARIOLIFORMIS (Hebra-Kaposi), *Acné pilaire* (Bazin), *Acne with depressed cicatrices* (Besiner-Doyon), *Acne frontalis or necrotica* (Boeckh), *Acne rodens* (Leloir-Vidal), *Folliculitis varioliformis* (Unna), is an eruption usually of adults, affecting the borders of the scalp, particularly the forehead, or involving the scalp, the whole face, the upper part of the chest, or the back; and probably on rare occasions still more extensive. It runs a chronic course by the constant evolution of fresh lesions, and it may recur. It is characterised by the formation of rosy projections seated around the pilo-sebaceous orifices, or of shotty acneiform nodules, or occasionally still larger nodosities. These papules may, or may not, become capped with a degree of pustulation (sometimes umbilicate), and it is characteristic of them that they undergo a necrotic mummification, so that the crust forming, at first yellowish and later darker, rapidly sinks into the skin and accurately covers an indelible cicatrix, shallow or deep and varioliform, which is finally disclosed by the fall of the crust. No comedones are formed. The eruption is usually disseminated, but it is also described as forming linear, more or less circinate and corymbose groups. All degrees of severity are seen, depending on the number and size of the lesions, the intensity of the process, and the regions involved. Unna separates Boeckh's closely related acne frontalis necrotica.

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[1] These bacilli are $\frac{1}{3}$ to $\frac{1}{2}$ μ broad by $1\frac{1}{4}$ to $1\frac{1}{2}$ μ long, or half that length, and many show a constriction or unstained cross-line in the centre. They are arranged like irregular bundles of spelicans, readily stained with basic aniline dyes, if not too old, and are completely decolorised by acids.

UNNA ascribes the distension of the follicle, and its closure by a firm scale of horny lamellæ, to the enormous growth of a small bacillus within it, morphologically and tinctorially very similar to his acne bacillus, but without the same tendency to longer growth. Crust formation, he says, is due to the secondary growth of the diplococci of seborrhœic eczema, which supplant the bacilli, and, finding themselves in a peculiarly favourable soil, set up a sero-fibrinous inflammation of a character unusually deep and intense for them, and necrosis often results.

With a little experience the *diagnosis* is generally easy. The acneiform papulation and pustulation without comedones, the peculiar necrosis, the depressed crusts, the indelible varioliform scars with the special localisation are very significant. Recurrent localised syphilides give rise to the greatest difficulty. Acneiform lesions with necrosis may also occur in scrofulo-tuberculous subjects ("tuberculides"), but the border of the scalp is a very exceptional site for them.

ACNE ROSACEA, or, better, *Rosacea*, is the term given to a chronic affection in adults of the glabrous portions of the face, sometimes extending to the neck, characterised by the presence of one, or generally a combination of several, of the following changes—(1) Hyperæmia, smooth, superficial, or deep, at first intermittent, then remittent, and lastly permanent with exacerbations; (2) hyperæmia or catarrh with some exfoliation or increased outflow of oil, and often associated with "eczema seborrhœicum" of the scalp; (3) profound erythema with infiltration of the corium of various degrees, at first œdematous, then plastic, with either comparatively few inflammatory papules or papulo-pustules, or with a severe red, coppery, or vinous acne (nodular and pustular); (4) telangiectases or dilated veins of various sizes, in lines or networks, localised or diffused; (5) in extreme or very chronic cases hypertrophic changes leading to a very general thickening of the tissues of the face. An extreme form of the latter change, localised to the nose and immediately adjacent portions of the face, is known as *rhinophyma*, and consists of a voluminous tumour-like hypertrophy of the sebaceous glands, or of a fibromatous growth, or of both in various combinations.

The essential basis of rosacea appears to be either a hyperæmia or a seborrhœic catarrh of the skin, or a combination of them in a hyperæmia which favours the determination of a seborrhœic catarrh. The other changes are consecutive or secondary. According to Unna, rosacea in nineteen cases out of twenty is a seborrhœic catarrh, secondary to a similar affection of the scalp. There is, however, a special tendency to vessel dilatation and an absence of the usual epithelial growth and spongy metamorphosis. Like any other seborrhœic catarrh rosacea may be conjoined with secondary purulent infections and furuncles may develop. In the twentieth case rosacea is a simple angio-neurosis which never leads to hypertrophy. We think that Unna does not bring into sufficient prominence the hyperæmia of the face, which is really the essential basis, and on which the seborrhœic catarrh and secondary infections may be engrafted. This hyperæmia is sometimes directly produced, as in cooks and others exposed to the fire, or in coachmen exposed to the weather; frequently reflex from catarrh of the stomach, or from disorder of the genital apparatus, and then intensified at the menstrual periods or at the menopause. Alcohol is a frequent cause of the stomach catarrh.

Rosacea is thus apt to be a somewhat complex affection, and its manifestations range through the various degrees of more or less stereotyped hyperæmia, generally with the added phases of seborrhœic catarrh, and secondary infections with pustulation. It may affect the whole flush area of the face or only certain portions. Some difficulties of *diagnosis* may arise from late suppurating nodular syphilides localised to the face, and the rare diffuse hypertrophic forms may simulate the leonine aspect of leprosy.

TREATMENT.—As the etiology of acne vulgaris is unsettled, the treatment is to some extent empirical.

Internal Treatment.—Whatever the influence of gastro-intestinal or genital disorders, or general states, such as anæmia and poor nutrition with feeble circulation, may be, it is unwise to neglect the correction of any departure from the standard of health. Any defect in this direction should be corrected by appropriate means which we need not consider in detail. There are no specific internal remedies on which we can rely. Arsenic has no great influence. Sulphide of calcium was at one time highly recommended in pustular acne, but is disappointing. Ergot also has its advocates, especially where menstruation is at fault.

Local treatment is of most importance in the removal of the eruption, and our endeavours must be directed (1) to clear away all existing lesions, and (2) to prevent the formation of fresh ones. In patients pursuing their daily avocations the active treatment about to be discussed should be carried out at night, and soothing or cosmetic remedies, such as cold cream or the calamine lotion to which bichloride of mercury may be added, applied in the daytime.

The comedones plugging the pilo-sebaceous follicles are the basis upon which so many secondary changes are grafted, and it is therefore absolutely essential to clear them all away with the contained lanugo-hairs and organisms.

The comedones are extracted by means of the finger-nails, guarded by a silk handkerchief, or by a watch-key with bevelled edges, or by one of the numerous comedo-extractors recommended. The extraction is greatly facilitated by loosening the plugs by means of a pricker or needle, and particularly by a preliminary softening of the skin by prolonged steaming, bathing with hot water, or a local water bath effected by means of an impermeable dressing. A vigorous shampooing or friction with a towel is desirable. Massage has also been recommended, and Nevins Hyde has devised a "massering ball" with which he rolls out the plugs. Extraction of comedones should be immediately followed by the application of some disinfectant lotion, or, better still, ointment.

Not only is it difficult, however, in many cases to extract all the comedones in this way, but fresh ones continue to form unceasingly. Moreover, we have to deal with the generalised changes in the skin, which Sabouraud ascribes to a parasitic oily seborrhœa, and Unna to a hyperkeratosis. Whether we adopt either of the latter theories or fall back on the older view of a purely functional disorder, it is found practically that the local application of certain remedies with keratolytic, stimulating, reducing, and disinfectant properties are useful. Soap is constantly interdicted, nevertheless a *soap treatment*, and often a vigorous one, is apt to be most useful. The selection of a soap, and the energy and frequency of its application, must depend on the case and the reaction. The soap may be left on all night, or be washed off, and the skin then soothed by a bland powder, or be further treated. Sometimes toilet, soda, or castille soaps suffice, but generally a soft potash soap is called for, such as the lin. saponis, or lin. potass. iodidi c. sapone, or Hebra's sp. saponis alkalinus (sap. virid. ℥ij, sp. vini rectif. ℥j). A sand soap (Auspitz) or milder marble soap (Unna) finds favour with some. Amongst medicated soaps (superfatted or alkaline, powder, liquid, or solid) those containing sulphur, ichthyol, salicylic acid, naphthol, or bichloride of mercury are useful. The methodical application of these soaps causes a maceration and stimulation of the skin, a moderate exfoliation of the cuticle and lining of the follicles, and helps the disinfection. To assist these processes we can apply, after removal of the soap, or at other times, one of the following lotions. The type of the sulphur lotion is Kummerfeld's *lotio sulphuris cum tragacanth* :—

Sulph. præcip.	ʒij.
Camphor	gr. x.
Pulv. Tragacanth	ʒj.
Aquæ Calcis et Aquæ Rosæ	āā ℥ij.

This formula is varied endlessly, chiefly by the substitution of ether and spirit for the lime and rose-waters, and glycerine for the tragacanth, or the latter ingredient is omitted. Vlemingkx's solution (quicklime, ʒss; sublimed sulphur, ℥j; water, ℥x: boil down in an iron vessel to ℥vj, and filter) is too strong, and must be well diluted and used carefully. Sulphurated potash may be used in the following formulæ :—

Potas. Sulphurat.	ʒj.
Tinct. Benzoin	ʒj.
Glycerine	ʒiss.
Aquæ Rosae ad	℥iv.

Kaposi gives the following *pastes:* lac sulphuris, 10; spirits of wine, 10; glycerine, 1·50; or sulphur, 10; spirit of soft soap, 20; spirit of lavender, 60; balsam of Peru, 1·50; spirit of camphor, 1; oil of Bergamot, 5 drops. Unna's *pasta zinci sulphurata* consists of equal parts of oxide of zinc, precipitated sulphur, terra silicea, and benzoated lard. There are very few better preparations than *Wilson's hypochloride of sulphur ointment* :—

Sulphuris Hyperchlor.	ʒss-ʒij.
Potasii Carbonatis	gr. x.
Adepis Benzoat ad	℥j.

Perchloride of mercury lotions are cleanly, but the possibility of poisoning by prolonged use must be kept in mind, and its incompatibilty with sulphur.

In obstinate cases, and where the patient can give himself up to the treatment, *peeling pastes* have been used in recent years, for instance, Lassar recommends β naphthol, 10·0; precipitated sulphur, 50·0; vaseline and green soap, of each 20-25·0, to be spread on, and wiped off with a soft rag after fifteen or twenty minutes, and then the skin powdered with talc. After two or three applications some inflammation is set up and the horny layers peel off. A 50 per cent resorcin paste is also effective. Unna makes use of an acetic acid paste, for example, terra silicea, 10·0; glycerine, 25·0; concentrated acetic acid and resorcin, of each 2·5.

Where *suppurating lesions* exist, the pus—and a bead is often embedded in a solid-looking lesion—must be let out by a bistoury, lancet, tenotomy knife, etc., and the incised parts sterilised by a parasiticide. For the obstinate special lesions, called acne indurata, free incision or multiple puncturing is practised, or the application of a drop of pure liquid carbolic acid, followed by a painting with flexile collodion. They sometimes yield to biniodide of mercury ointment (grs. v-xx ad ℥j), and also to the application of resolvent plasters, such as emplastrum hydrarg. comp.

In severe *A. pustulosa* and *indurata* cases, excellent results are claimed from the operation of free curetting and scarification, but I have rarely found this necessary. The belladonna ointment recommended by Stephen Mackenzie is certainly most useful for the larger inflammatory nodosites seen in some cases, as on the back.

Acne varioliformis, in my experience, is largely controlled by the internal administration of arsenic. The treatment usually relied on is an external one on much the same lines as that for acne. The skin and follicles are kept disinfected as far as possible, and resolvents, in the shape of mercurial or sulphur ointments and pastes, are most in favour.

In *Rosacea* the causes of the hyperæmia of the face must be determined and dealt with. If direct, as in those exposed to fire heat and the changes of the weather, they must be prevented as far as possible. If of gastro-intestinal origin, such disorder must be carefully defined and treated accordingly, dietetically and medicinally. Frequently a degree of gastritis exists, and bismuth is one of the most commonly useful medicines. The alcoholic cases are unsatisfactory, as the cause is apt to be constantly in operation. Reflex hyperæmia from disorders of the genital apparatus is also likely to prove troublesome, especially when dependent on some chronic trouble, as ovaritis or metritis, or when occurring in association with the menopause. The range of treatment is also obviously wide. In still another series of cases the hyperæmia appears to be due to an excitable and unstable state of the vaso-motor system of the face, so that the face flushes under very slight stimuli. In addition to the specific measures indicated for the relief of local gastro-intestinal and genital trouble, we seek to diminish the reflex excitability by such medicines as bromide of potassium, and to control the hyperæmia by such drugs as ergot and the proprietary sulphur compound known as ichthyol. A course of the latter is often of value, and salicin also promises to be useful. The cyanotic conditions, sometimes localised to the nose, arising from a feeble circulation, are not so apt to be associated with the inflammatory elements, and are difficult to control. Every effort, however, should be made in the way of local stimulation and the promotion of the general nutrition and circulation.

The local treatment is directed to (1) the control of hyperæmia; (2) the subduing of inflammation; (3) the management of pus formation; (4) the obliteration of dilated veins. Where hyperæmia is a prominent symptom, cooling, evaporating, and soothing astringent lotions, such as lotio calaminaris, and lead and spirit lotion (if sulphur is not also used), are called for, and are also useful as a day application when more active treatment is carried out at night. Inflammation, such as seborrhœic catarrh, must be controlled by sulphur, resorcin, salicylic acid, ichthyol, or mercurial lotions, ointments, or pastes, such as are recommended in acne vulgaris. Pustules should be opened and fomented with hot boric acid lotions. In inveterate cases with considerable thickening it is sometimes desirable to scarify areas or to curette inflammatory lesions. Telangiectases are easily destroyed by electrolysis, or more roughly by incising them.

There is a form which must be included with rosacea, in which large papulo-pustules recur on some portion of the skin, such as the chin or one cheek, or the nose, independently of any hyperæmia. The condition is much intensified at the menstrual epochs, and is often extremely obstinate and annoying.

The tumour formation of the nose, known as *Rhinophyma*, demands surgical treatment by ablation or decortication and subsequent grafting by Thiersch's method. Slighter forms may be treated by scarification or by Lassar's method of multiple puncturing. He mounts forty points on a solid disc one centimetre in diameter, and works it by a dentist's filling electromotor.

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Aconite. *See* TOXICOLOGY.

Acrodynia. *See* PELLAGRA.

Acromegaly (Pachyakry, v. Recklinghausen).—DEFINITION AND HISTORY.—Acromegaly is a disease characterised by an enlargement due to hypertrophy of the terminal parts of the body, the hands and feet, and the head, including the face, being principally affected.

Marie in 1886 was the first to recognise the entity of this affection, cases of which had been described before under various names, but mostly as forms of giant-growth. A typical case was described by Cunningham in 1879. The patient suffered from diabetes, and a tumour of hypophyses was found after death; another case was described by Hadden and Ballam in 1885.

Since the publication of Marie's paper numerous cases have been recorded, amounting now to over 240. Marie separated from acromegaly an affection secondary to chronic pulmonary disease, in which an enlargement of the joints (wrists, ankles, knees), with clubbing of the fingers and toes, is found, and to which he gave the name of osteo-arthropathie hypertrophiante pneumonique (*see* "Osteo-arthropathy"); and more recently Marie distinguishes two types of acromegaly—"type en large," in which there is chiefly increase of thickness of the hands, and "type en long," in which their length is increased. Brissaud and Milignan describe under acromegalo-gigantum a combination of acromegaly with giant-growth. Combinations of acromegaly with myxœdema, with syringomyelia, and with Graves' disease have also been noticed.

ETIOLOGY.—The disease occurs in all parts of the world. Owing to its very insidious onset it is difficult to determine the exact age at which it commences; in most cases the symptoms are fully developed between the ages of twenty and forty, some cases have been described of an earlier age (age of puberty), others as commencing at a late period (forty to fifty). It occurs more frequently in women than in men. Heredity has been noticed in two cases. Of other etiological factors we may mention: acute infectious diseases, fright, shock, traumatism, alcohol, rheumatism, and syphilis. In most cases, however, no satisfactory cause can be made out.

SYMPTOMS.—When the disease is fully developed the appearance of the patient is very characteristic. The head is larger, and the increase affects the bony and soft parts. The skull is larger, and the occipital protuberance stands out markedly. The face is enlarged and oval, the orbits and zygomatic arch prominent, the nose is increased in all directions, the septum forming a broad bar, but most prominent is the size and projection (prognathus) of the lower jaw; the lower lip is prominent, the ears are often very large, and the cartilage of the ear is thickened. The hands are enlarged, but keep their natural outline, as the increase affects both the soft and bony structures, the fingers are broad like sausages, the nails rather small, the feet are large, the toes broad, especially the big toe. The skiagraph shows the enlargement to be due chiefly to the soft parts, the bones are only slightly enlarged, and show occasionally a few osteophytic deposits, the forearm is often increased in thickness, the rest of the extremities not markedly affected. The vertebral column in fully developed cases shows pronounced kyphosis in the cervical region, and all the bones of the thorax show an increase in size; the clavicles are broad and stand out, and the lower part of the sternum shows a marked prominence.

The appearance of a patient suffering from acromegaly is thus very characteristic, and the affection may often be recognised at a glance. Of other symptoms we may mention—

General stature.—It is noticed that many of the patients are above normal size (20 per cent, according to Sternberg); *the skin* and *subcutaneous tissue* is found thickened over the affected parts, but not otherwise altered, so that there is a marked similarity of appearance and facial expression in the different patients, the nails are small, the hair shows no marked change, the perspiration is often increased. Occasionally the skin is like that of myxœdema.

Respiratory system.—One often notices an alteration of voice, which becomes thick or husky, and this is due to an enlargement of the cartilages of the larynx and thickness of the mucous membrane; patients often suffer from attacks of dyspnœa, partly due to rigidity of the thoracic walls, and partly due to weakened heart action. In many cases (mostly in women) an enlarged *thyroid* is noticed, in others the thyroid appears diminished. In a good many cases marked dulness of percussion over the sternum has been noticed, and this has been put down to the persistent enlargement of the thymus (Erb); in several post-mortem examinations (one of my own cases) a quantity of fat was found in the anterior mediastinum, but scarcely any glandular structure.

The *circulatory* system shows in most cases, when the disease has existed for some time, distinct dilatation and weakness of the heart.

Digestive tract.—Tongue very much enlarged, the mucous membrane and the lymphatic structures in the pharynx thickened, disturbances in the functions of the digestive tract, such as increased thirst, increased appetite, constipation, etc., are often noticed.

The *liver* and *spleen* are often enlarged, also the *lymphatic* glands, superficial and deep.

Urine.—In many cases sugar appears in the urine, sometimes only in small quantities and intermittently; in others pronounced diabetes may occur, and death from diabetic coma is not rare (Bury, Cunningham, etc.); in others, one has noticed diabetes insipidus (this has been described in tumours of the hypophysis, even when there was no acromegaly); albuminuria occurs frequently.

Generative organs.—The penis, prepuce, labia, and clitoris are often enlarged; with the development of the disease amenorrhœa occurs (Sternberg).

Nervous system.—Most of the symptoms are of functional nature, yet muscular atrophy of the type of progressive muscular atrophy has been noticed, sometimes only atrophy of a few groups of muscles; on the other hand, headaches, occasionally of very severe and persistent nature, vertigo, pain, and numbness in the extremities are very common symptoms; the patient often shows signs of mental depression, signs of neurasthenia, and occasionally mental aberration.

The reflexes are generally normal, only rarely are they absent (Freund).

Interesting are the changes relating to the eye. A tumour of the hypophysis being so often found in this disease, the optic nerves in their course are compressed either in or near the chiasma, and thus changes in the retina (atrophy) and disturbance of vision have often been noticed, the latter often giving an indication as to the situation of the tumour; thus in a few rare cases bi-temporal hemianopsia has been noticed, in others homonymous hemianopsia with hemiopic reaction of the pupil; whilst in others either blindness from atrophy of both papillæ or blindness in one eye and hemianopsia, or contracted field of vision of the other eye, form the principal symptoms.

Other affections relating to the eye which have been noticed are thickening of the lids and tarsus, hypertrophy of the lachrymal glands and increased secretion of tears, exophthalmos either of one or of both eyes, paralysis of one or more ocular muscles.

Affections of smell and taste have been noticed, and also deafness, probably due to diminution of the lumen of the meatus (Sternberg).

Course and Termination.—The disease runs a more or less chronic course; it commences insidiously, and beyond slight pains and sensory disturbances the patient only notices that the feet and hands and head get larger, and that he requires a larger-sized boot, glove, or hat. In some cases the affection of the eyes arouses the patient's attention. The symptoms develop slowly, and sometimes the condition remains stationary for a time, and then follows a fresh recrudescence; eventually the patient becomes weak, is obliged to take to his bed, and death takes place from exhaustion or dilatation of the heart. In other cases death may occur suddenly either from diabetic coma or from the symptoms of cerebral tumour; the duration of the disease is in most cases ten to twenty years, or even more.

In some few cases the course has been a shorter one (malignant form of

Sternberg), yet it lasts several years; such cases are described by Caton, Hansemann, Uthoff, Rolleston. The enlargement of the extremities takes place more quickly, and death takes place more or less suddenly; in all the acute cases a sarcomatous tumour of the hypophysis has been found.

Pathological Anatomy—Epidermis.—This is a true hypertrophy, the cells of the rete Malpighi contain pigment, the cutis is increased, the sheath of the nerves, the walls of blood-vessels are thickened, the fibrous tissue is firm and sclerotic.

The *muscles* for a long time remain unaltered; towards the last they show marked atrophy of the muscular elements and increase of the fibrous tissue.

The *cartilages* are thickened, showing proliferation of the cells and deposits of fibrous tissue; the cartilage covering the end of bone entering a joint is either thickened or thinned, and may be the seat of osteophytic deposits.

The changes in the *bones* have been carefully studied by several observers (Arnold, Schultze, Sternberg, Hunter), and have not always been found to be of the same nature; hypertrophy, osteophytic growths, and absorption of bone are noticed. The periosteum is often found thickened, and leads to the apposition of new layers of bone, as in the bones of the skull, and from it also proceed osteophytic deposits, and ossification of the insertion of muscles and fasciæ. The medulla of the bone is also the starting-point of further changes, and may lead to new formation of bone or to an absorption of bone.

Hunter found great vascularity of the medulla of bone and hæmorrhages. The skull is enlarged in its circumference and thickened, and the outer surface roughened where muscles and fasciæ are inserted; the foramina for the vessels are enlarged, and osteophytic deposits on the inside of the skull are noticed. The hypertrophy of the bones at the base of the skull causes often a narrowing of the foramina for the cranial nerves; in the numerous cases in which a tumour of the hypophysis has been found the sella turcica has been found enlarged in all directions, and the bones in the neighbourhood may show atrophy or are displaced.

The lower jaw shows great hypertrophy both of the body and rami.

The kyphosis of the cervical spine is due in some cases to the bodies being smaller in front than behind. The vertebræ have their anterior surface roughened, and are the seat of osteophytes.

The ribs are compact and increased in size, likewise the sternum, and especially the clavicle.

The long bones of the extremities show chiefly osteophytes, roughness of the muscle-insertions, and enlargement of the tubercles and condyles; the same is noticed in the bones constituting the carpus and tarsus, the metatarsal and metacarpal bones, and are somewhat broader and have small exostoses on the palmar and plantar surface; the phalanges show thickening of the epiphyses and osteophytes.

The *heart* has been found hypertrophied and dilated, the blood-vessels show thickening of the intima and adventitia, and degeneration of the muscular coat, and these changes can be traced to the smaller vessels.

Of the *internal organs* some show marked enlargement, such as the spleen, liver, kidney, lymphatic glands, but the enlargement is not always due to hypertrophy. The stomach and intestine are often enormously enlarged, and in one case the intestine was twice the normal length (Sternberg).

The pancreas, in some cases in which there was diabetes, was found fibrous, and other changes, such as necrosis, have been noticed.

Of the *external genital* organs we have already spoken. The uterus has been found in an infantile condition in several cases, with the ovaries in a state of cystic degeneration. The thyroid gland may be either atrophied or enlarged, or the seat of cysts, or affected with colloid degeneration.

Nervous system.—Thickening of the peripheral nerves, and of cranial nerves, and of the nerve-roots with neurotic change, has been noticed, also enlargement of the spinal ganglia, due to sclerosis, also various changes in the spinal cord (sclerosis of the posterior column); the brain has been found hypertrophied by Klebs, but the most noteworthy effect is seen in the hypophysis. We may say that in all cases of acromegaly in which a post-mortem was made, about thirty-six in number, the hypophysis or a part of it was found affected. In most cases a tumour was found to spring from and replace this organ. The nature of the growth varied a good deal, in some cases simply hypertrophy of the pituitary body was found, with or without colloid degeneration, in others an adenomatous growth; whilst in others the tumour was of a sarcomatous or gliomatous nature; the tumour by its growth compresses the neighbouring parts; pressure on the chiasma may cause atrophy of the optic nerves, or by backward extension affects the oculo-motor nerve and the cerebral peduncles.

Pathogenesis.—As yet it cannot be said that the pathology of acromegaly has been satisfactorily made out, though several views have been expressed to explain the causation of this peculiar and so well defined disease. Briefly enumerated these are:—

1. The neurotic theory. Recklinghausen and others have looked upon acromegaly as a neurotrophic affection. This view is based on the fact that in some observations changes in the nervous system, especially in the sympathetic ganglia and in the peripheral nerves, have been noticed. In many cases no noteworthy changes have been found in these nervous structures, whilst the trophic changes seen in nervous affections are generally of a different nature.

2. Klebs believed that the disease was due to the persistence of the thymus gland. This theory is no longer tenable, for in many cases of acromegaly the thymus gland was either absent or had undergone fatty changes. Furnivall's analysis shows that out of nineteen fatal cases it was absent in seven, hypertrophied in three, persistent in eight.

3. Some believe that to alterations in the thyroid gland, which had been found enlarged in some cases, must be ascribed the primary cause of the disease, especially also as occasionally symptoms of myxœdema are noticed, but Furnivall's table shows that out of twenty-nine cases in which the condition of the thyroid gland was observed, it was found normal in five, hypertrophied in seventeen, and degenerated or atrophied in the rest of the cases.

4. Purely theoretical is the view held by some that the disease is due to an involution, a return to the type of the anthropoid ape.

5. Many now hold the view that the changes in pituitary body may explain the symptoms. In favour of this view it must be stated that in all the fatal cases (Furnivall gives thirty-four, to which I may add three more, one of my own not yet published) the pituitary gland was found affected.

This gland, complex in its structure, occupies a peculiar position, both as regards its development and probable function. It exercises a trophic influence on the nervous system, and its hypertrophy, which was noticed in so many of the fatal cases of acromegaly, might explain some of the symptoms of acromegaly. According to this theory the pituitary body stands in the same relation to acromegaly as the thyroid gland to myxœdema, or more likely to Graves' disease.

Against this view may, however, be cited—

1. That tumours of pituitary gland have been noticed without any symptoms of acromegaly.

2. The changes in the pituitary body found in acromegaly are so very different, and

3. That the hypertrophy of the pituitary gland often noticed in the post-mortem cases examined may merely be a symptom and part of the general hypertrophy.

Differential Diagnosis.—The diagnosis of the affection when fully developed is easy enough; in its earlier stages the symptoms of cerebral tumour (atrophy of discs, vomiting, headache) may preponderate, or in the acute case the symptoms of diabetes.

Affections which it may simulate are—

1. Myxœdema. In this affection there is noticed slowness of speech,

tremors, bland and apathetic expression of face, the coarse character and scaly appearance of the skin of the hands, which are large and spadish, but the thickening is due to the soft parts and not to the bone. There is no prognathus and no marked increase in size of the head.

2. In Paget's disease—here we may have enlargement of the head, kyphosis, thickening of the bones; the deformities, however, of the long bones in this disease, and the absence of the thickening of the soft parts, form sufficient differential points. Moreover, the hands and feet are not affected, the lower jaw does not project, and the bones of the face not enlarged.

3. Diffuse hyperostosis, and the various types of giant-growth (Sternberg).

TREATMENT.—Removal of the tumour of the pituitary body has been attempted in one case (Caton), but unsuccessfully, and trephining of the skull to relieve intracranial pressure (Lynn Thomas). The organic extracts, thyroid gland extract, and extract of pituitary body, either the one or the other, or both combined, have been extensively tried, but with varying results, and as many cases of acromegaly show often a temporary recession in their symptoms, it is difficult to say how much of the improvement noticed is due to the treatment. Iodine and iodides have been given for some time, and in some cases with distinct amelioration of the symptoms. Cases complicated with diabetes require, of course, careful dietetic treatment, and large doses of alkalies may prevent the occurrence of diabetic coma. Massage and electrical treatment have given only very equivocal results.

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Actinomycosis.

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Actinomycotic Lesions—

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DEFINITION.—Actinomycosis is an infectious disease due to the penetration into the tissues of a vegetable parasite, the actinomyces or ray fungus.

General Description.—It has a chronic, progressive course which, when unchecked, tends to a fatal issue. Its symptoms are generally obscure, but not infrequently resemble those of chronic tuberculosis. It is characterised by the formation of granulomata, which, in man, usually take the form of multiple small abscesses surrounded by a considerable amount of granulation tissue. The tendency of these lesions is to invade neighbouring tissues in one or more directions; the oldest parts of the lesions not infrequently heal by cicatrisation. Besides man, in whom the disease is comparatively rare, many omnivorous and herbivorous animals (especially cattle) are liable to actinomycosis. The disease has been chiefly observed in the temperate zones; it is contagious, but as a rule infection appears to be conveyed by plants, and more specially by some of the gramineæ. There is much evidence showing that the disease may be cured by the administration of large doses of iodide of potassium.

Allied Diseases (*Affinities*).—Actinomycosis has many pathological features in common with mycetoma (Madura foot), botryomycosis, tuberculosis, syphilis, it has more distant affinities to Farcin de bœuf, glanders, and chronic pyæmia. According to a view, which has not been generally accepted, mycetoma would be a variety of actinomycosis.

History.—The disease was first duly recognised in cattle by Bollinger (1876); he submitted the parasite to the botanist Harz, who named it the actinomyces or ray fungus (1878). Israël soon afterwards described the disease in man (1877), and in 1879 Ponfick showed the identity of the human and bovine disease.

Johne (1880) was apparently the first to prove that the disease was inoculable. In 1883 Israël succeeded in transmitting it from man to rabbit, and several years later Wolff and Israël succeeded in producing the disease by the introduction of pure cultures of the actinomyces into the peritoneal cavity of rabbits and guinea-pigs.

In 1885 Boström cultivated successfully the parasite, and gave reasons for believing that it belonged to the cladotrichæ and not to the mould-fungi as Harz had been inclined to believe.

In 1885 also Thomassen announced that actinomycosis could be cured by the administration of large doses of iodide of potassium.

In England, Fleming (1882) was the first to attract attention to the existence of the disease in cattle, Mr. Knight Treves the first to describe a case of human actinomycosis (1884). Acland shortly after established fully the existence of the disease in the country.

Although of recent discovery, actinomycosis is not a new disease; as far back as 1826 Leblanc gave a good description of actinomycotic lesions of the jaw in cattle (under the name of osteosarcoma). Perroncito and Rivolta (1868) had even noticed the presence of the parasite in the same kind of lesion, and partly surmised its nature. Hahn in 1870 made similar observation in connection with the "wooden" tongue.

With regard to human actinomycosis, Langenbeck in 1845 observed actinomyces granules in pus obtained from a case of vertebral caries; he, however, did not at the time realise the importance of that observation, and his case was published only in 1878 by Israël. The first case actually published was that recorded by Lebert in 1848; this observer showed and depicted very accurately the actinomyces granules, which he found in pus coming from a thoracic abscess, but he did not recognise their vegetable nature.

Synonymy.—Actinomycotic lesions in man have undoubtedly been repeatedly taken for chronic abscesses, tumours, chronic tuberculous, or syphilitic lesions.

In cattle analogous mistakes have been made, both in this country and abroad, for these lesions have been and are still frequently described under the names of scrofulous, strumous, or tuberculous abscesses or inflammations, bone tubercle, cancer of bone, cancer of tongue, scirrhus cord, scirrhus tongue, osteosarcoma, spina ventosa, not to mention a number of less scientific but more correct descriptive terms.

Clinical Features and Diagnosis.—There is no special symptom by which actinomycosis can be surely recognised. It has usually a chronic course, and according to the part of the body affected the symptoms may be those of tuberculous caries of jaws, vertebræ, or ribs, of chronic abscesses of various kinds, of empyema, sub-phrenic abscess, peritonitis, chronic phthisis, ulceration of the bowel, tumours of bone or of viscera, etc. Fever may be present or absent. The disease occasionally assumes an acute form, owing to vascular infection, and the symptoms may resemble those of pyæmia; in such cases the possibility of a mixed infection should be kept in mind. A reliable diagnosis can seldom be made before some of the abscesses have opened upon the surface of the body, or into some cavity opening externally; the characters of the discharge may then suggest the presence of the actinomyces. The discharge may be viscid, lumpy, or resemble closely laudable pus; when spread in thin layers it is seen to contain a large number of minute whitish or yellowish specks just visible to the naked eye; on microscopical examination these specks present the microscopical features described further on.

Distribution of Lesions.—All the tissues, and probably all the organs of the body, are liable to the disease. Lesions have been described in the following parts: mucous membranes (alimentary, respiratory, and genito-urinary tracts), serous membranes (pleura, peritoneum, etc.), skin, bone, muscle, lymphatic glands, lungs, heart, liver, spleen, kidney, prostate, ovary, mamma, brain, etc. The characters and the distribution of the lesions depend in great part on the mode of infection. Israël recognised three of these—(1) by the mouth, (2) by the lungs, (3) by the intestine, to which we must now add (4) by the skin, (5) by the genital tract. Their relative importance is well shown by the cases collected by various observers, and notably by Illich. It will be sufficient to refer here to the figures given by Leith, for they cover the cases previously published by Illich, and some thirty additional ones.

	Cases.	Per cent.
Actinomycosis of the head and neck	224	55·75
„ „ tongue .	16	
„ „ abdomen .	93	21·60
„ „ lungs . .	57	13·25
„ „ skin . .	11	2·50
„ „ doubtful .	29	6·90
	430	100·00

In addition to these, the cases reported by Zemann and Grainger Stewart and Muir indicate the possibility of infection through the vagina.

Primary Actinomycosis of the Mouth and Pharynx.—One or more teeth are generally found in a state of caries, or there may be wounds or ulcers affecting the mucous membrane of the tongue, gums, tonsils, or pharynx. These lesions are generally supposed to favour the penetration of the parasite. The most frequent result of infection through the mouth is affection of the lower jaw, but the tongue, the upper jaw, the bones of the skull, and the vertebræ may also become involved; the disease may in this way extend to the cranial cavity and to the spinal canal.

Actinomycosis of Bone.—In man the tendency of this disease is to produce progressive necrosis, suppuration, and periostitis. In cattle the same processes may occur, but are generally overshadowed by extensive connective tissue hyperplasia, which gives rise to the formation of large tumours resembling closely osteosarcomata.

In man the lesions of the lower maxilla affect chiefly the periosteum and surrounding tissues; they may take the form of burrowing abscesses or of small tumours situated on the surface of the jaw or in the sub-maxillary region; they are often connected by fistulous channels with the root of a carious tooth. As the suppuration progresses the skin may become involved. Soltman's case indicates one of the ways in which the vertebræ may become affected, and is also of great interest in showing the mode of infection. The patient, a boy, had swallowed an ear of wall-barley (*Hordeum murinum*), a spikelet of which penetrated the walls of the œsophagus. This led to the formation of an actinomycotic abscess, which after a time pointed through the sixth intercostal space, between the spine and scapula.

Actinomycosis of the Tongue is of chief interest in connection with cattle, where it is known as the "wooden" tongue (also tubercle, chronic sarcoma, chronic interstitial glossitis, etc.). The state is characterised by the formation of actinomycotic nodules under the mucous membrane which, after a time, becomes ulcerated over the parts affected; a considerable increase of connective tissue in the deeper parts of the organs (interstitial myositis) gives rise to atrophy of the muscular fibres and great hardness of the organ.

Actinomycosis of the Intestine.—The large is more frequently affected than the small intestine. The lesions may consist of small abscesses spreading chiefly in the submucous coat. These may point towards the surface of the mucosa, discharge into the cavity of the intestine, and their contents appear in the stools. In a case recorded by Chiari the mucous membrane presented numerous raised patches, or nodules, around which the membrane was swollen and congested. Softening of these nodules gave rise to ulcers with undermined edges. Cicatrisation of intestinal ulcers may take place.

After a time the peritoneum becomes involved, adhesions are produced, the abscesses may spread in various directions; they have the tendency to open externally. In some cases they have been found to discharge into the bladder, or to involve the prostate, liver, etc. In the case recorded by Ransom, the abscess had extended from the rectum to the prostate, and actinomyces granules were found in the stools and in the urine of the patient.

Actinomycosis of the Liver.—The liver is usually affected by extension from the intestine, but sometimes it may become involved by abscesses extending from the lung through the diaphragm; and a case has been recorded in which actinomycosis of the liver had apparently been produced secondarily to abscesses of the skin in the neighbourhood of the umbilicus. These hepatic abscesses are variable in size, sometimes very large, situated more or less deeply, they have a typical alveolated structure; in their neighbourhood the parenchyma of the liver is deeply congested and in a state of atrophy, associated with intercellular cirrhosis. In the neighbourhood of these lesions the capsule of the organ is thickened, and there are usually peritonitic adhesions through which the abscesses may extend to neighbouring viscera or to the abdominal parietes.

Actinomycosis of the Lungs.—The lung may be primarily affected by inhalation of infected vegetable dust, or, secondarily, by extension of lesions of the skin, œsophagus, prevertebral tissue, liver, etc. There may be simply bronchitis and peribronchitis with some broncho-pneumonia, or the lesions may take the form of a chronic localised interstitial pneumonia and abscesses. These abscesses may be encapsulated. When the lung is invaded through the lymphatics or blood-vessels, actinomycotic nodules resembling miliary tubercles, but larger, may be produced. The pleura usually becomes involved, fibrous adhesions and empyema being the changes most commonly observed.

The disease has been diagnosed during life by the discovery of the parasite in the sputum. Abscesses starting from the lungs may involve any of the neighbouring organs, thoracic or abdominal, or extend towards the surface through the thoracic walls, and discharge externally. By spreading from the posterior mediastinum to the prevertebral lumbar region, these abscesses may simulate psoas or iliac abscesses.

Actinomycosis of the Skin.—The skin may be involved primarily by the penetration of infected splinters of wood, sharp appendages of the glumes of spikelets of barley or other grasses on which the organism may be present. It has already been shown that the skin is frequently involved secondarily.

The lesions appear as nodules, varying in size; these are sarcomatous-looking, soft, pulpy. They have a mottled, purplish red, and yellowish colour. The skin over them is thin; it ulcerates, giving rise to crateriform ulcers, which often coalesce and yield an abundant, clear, sticky discharge. Syphilitic-looking scars may be produced.

Actinomycosis of the Brain.—The brain is generally affected as a result of metastasis through the blood-vessels, but extension by contiguity following disease of the bones at the basis of the skull may also occur (Ponfick). These abscesses are generally multiple, and situated in the white matter at various distances from the surface of the organ; their size varies from that of a small pea to that of a small orange, the larger abscesses being subdivided into several smaller ones; but, generally speaking, the cerebral actinomycotic abscesses are larger than those found in other parts of the body. They seem to develop rapidly; in the case reported by Delépine abscesses reaching the size of a walnut had apparently been produced in not more than five or six weeks. Localised encephalitis and meningitis are observed round these abscesses. The nervous symptoms depend on the parts affected, but the supervention of paralytic or meningitic symptoms in a patient affected with buccal, intestinal, pulmonary, genital, or cutaneous actinomycosis should lead to a suspicion of actinomycosis of the brain and a bad prognosis, the patient seldom surviving more than a few weeks after the supervention of nervous symptoms.

Microscopical Anatomy.—A typical actinomycotic tumour or multilocular abscess, such as may be found in the human liver, is composed of a number of small, more or less rounded follicles of variable sizes, but seldom exceeding 3 mm. (⅛ inch) in diameter. Each one of these small foci may be described as an elementary abscess, follicule, granuloma, or shortly as an *elementary actynomicoma*. Near the margin of the tumour these elementary follicles are separated from each other by thick strands of very vascular and cellular connective tissue (granulation tissue), in the midst of which may be found atrophied, pigmented, and degenerated liver-cells; bile-ducts with proliferating epithelium; small arteries generally in a state of arteritis. In the more central parts of the multilocular abscess, the septa between the elementary abscesses are proportionally thinner, and they are entirely composed of granulation tissue, which is more or less degenerated and often pigmented; no distinct remains of the original tissues of the organ can usually be recognised in these septa. The primary abscesses are often grouped together much in the way as elementary miliary tubercles are grouped in chronic tubercles. The spaces separated by the septa are occupied by more or less degenerated leucocytes (pus corpuscles) which have migrated from neighbouring vessels; among them may be found more or less altered connective tissue corpuscles, epithelioid cells, or giant cells. In human lesions the leucocytes are usually so numerous that the elementary follicle has the characters of an abscess;

in cattle, the connective tissue elements predominate as a rule, so that the follicle has the characters of a solid granuloma, and resembles closely a miliary tubercle. About the centre of the follicle the parasite is usually found, it is closely invested by a layer of leucocytes or of epithelioid cells and giant cells. Many of the cells surrounding the actinomyces contain cocci (spores ?).

Characters of the Actinomyces.—Actinomyces grains vary in size; they may be granules, invisible to the naked eye, or be quite distinct and reach a diameter of 0·60 mm. or even 1 mm. ($\frac{1}{25}$ inch). The larger grains are usually brownish or brownish-yellow in colour, when seen by transmitted light; yellowish-white, sulphur-yellow, or, rarely, brownish, by reflected light. The small colonies are almost transparent and colourless. The granules found in human cases are usually soft and easily crushed; those from bovine cases are frequently gritty owing to calcification. These granules may be round, elongated or lobulated. Their structures may be ascertained by crushing or teasing them in a drop of water or saline solution (liquor potassæ or hydrochloric acid may also be used for special purposes). These simple methods are usually sufficient for diagnostic purposes, and the characters of the organism may be easily ascertained by means of a microscope, with a magnifying power of from 100 to 300 diameters. For a more complete study of the parasite, higher powers and various staining methods may be used, notably Gram's method, to bring out the filaments; staining by eosin, orange, or picrocarmin to study the clubs.

In an actinomyces grain, distinct to the naked eye, the following parts may be recognised from the centre to the periphery.

1. Interwoven filaments, measuring from 0·3 to 0·5 μ in diameter, branching and radiating towards the periphery. Many of the filaments are wavy, spirilla-like. Some contain rounded bodies resembling cocci or spores. Between the filaments, cocci, and short segments of filaments, resembling bacilli, are found. The cocci vary in size, but usually measure about 0·5 μ in diameter, they often appear in clumps in the centre of young colonies, and as previously shown are taken up by migratory and epithelioid cells.

2. The thickened claviform peripheral ends of the radiating filaments form a superficial layer, enclosing more or less completely the rest of the colony. These clubs are arranged radially, their thin end being directed towards the centre of the grain, they measure from 10 μ to 60 μ, or more, in length, and up to 10 μ across in their broadest part. These clubs may be branched. It is their arrangement which gives to the organism its typical appearance, and which suggested to Harz the name of Ray-fungus. In human lesions, specially when they are young or of rapid formation, the clubs may be absent, or small and inconspicuous. In cattle lesions they appear early, the filaments often become indistinct, and are difficult to demonstrate, specially after calcification has begun.

Cultivation of the Actinomyces.—Some of the grains are picked out of lesions freshly opened, free from adventitious contaminations; these grains are deposited on the surface of nutrient agar, glycerine agar, or potato, and in a few days, specially on glycerine agar, and at the temperature of the body, evidences of growth appear.[1] The actinomyces grows also in milk and other fluid media.

The growth on artificial media is composed of filaments, which become covered after a time with an efflorescence composed of cocci (spores ?); these cocci, when cultivated again, give rise to typical filaments. Such cultures may retain their vitality for over four years. The characters of the cultures of actinomyces granules are very similar to those of other organisms placed in the groups Cladothrix and Streptothrix.

Natural habitat.—Organisms similar to the actinomyces have been found on vegetable products, more specially on various parts of graminaceous plants. The actinomyces has not, so far, been cultivated from such sources, but Liebman states that when a grain is inoculated the parasite grows with the developing plant, which becomes infected in all its parts.

The sharp awns (bristles, barbs) of the floral leaves of the spikelets, composing the heads of gramineæ, would when infected be specially dangerous, so would also be stiff straw and splinters of wood. All these may easily penetrate the mucous membrane of the mouth and alimentary canal, and even the skin. Jensen in 1880 attracted attention to epizootics occurring in cattle fed on barley grown on newly cultivated land recently reclaimed from the sea. Vegetable debris have been found by various observers (Boström, Soltmann, etc.) in the midst of actinomycotic lesions, and sometimes forming the nucleus from which the parasite appeared to grow. Israël is of opinion that decayed teeth and the tonsillar crypts may constitute suitable places for the development of the fungus, but the leptothrix buccalis may simulate the arrangement of the actinomyces and lead to erroneous interpretations.

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[1] Optimum temperature, 32° to 37°; growth very slow at 18° C., arrested at 52° C. A temperature of 70° C. for ten minutes has been found sufficient to kill the organism.

Sources of Infection.—It is therefore probable that the chief source of infection must be found in infected grasses, and more specially in those of the genus *Hordeum*. There are very few cases indicating the possibility of contagion from cattle to cattle or from cattle to man. There is no evidence to show the occurrence of infection through the meat of diseased animals. The mamma may become affected in the human subject, in cattle, and in swine, and it has been surmised that the disease might be conveyed by milk, but there has been no record of cases showing that this form of infection had ever occurred.

There is no evidence to show that the disease has ever been inherited.

Experimental Actinomycosis.—Experimentally the inoculability of the disease has been fully demonstrated.

Actinomycotic pus obtained from human cases has been found to cause actinomycosis when introduced into the peritoneal cavity of rabbits, guinea-pigs, and calves. The guinea-pig and the calf are more readily infected than the rabbit; carnivora are generally refractory.

Transmissibility from cattle to cattle by inoculation has been demonstrated by Johne and by Ponfick.

Wolff and Israël have shown that cultures of the actinomyces grown outside the body may produce the disease when inoculated into the peritoneum of rabbits and guinea-pigs.

Occurrence and Prevalence—Geographical distribution.—Both in man and in the lower animals the disease seems to be most prevalent in Germany, Austria, Italy, Russia, and North America. Cattle are also frequently affected in certain parts of England. Actinomycosis has also been observed in France, Belgium, Holland, Australia, etc.

Distribution in the Animal Kingdom.—Bovidæ seem to be most liable to the disease. The statistics from various abattoirs on the Continent show that the proportion of cattle affected varies from 0·01 per cent to 2 per cent. In some herds in England 8 per cent of the animals have been found affected with various forms of actinomycosis (Crookshank). Swine and horses are also liable, but less than cattle. Cases have occurred in elephants, sheep, and even the dog (one case), but, generally speaking, so far as we know, the carnivora are practically immune.

In man the disease is comparatively rare, but it is not without practical importance. In the thirteen years from 1879 to 1892, about 450 cases have been published, and most of these have been recorded from 1886 to 1892.

Influence of sex and age.—Males are affected in the proportion of 73 and females of 27 per cent. The disease is most common between the ages of twenty and thirty, rare before ten and after sixty, but it may occur at any age.

Influence of occupation.—There is apparently no class or occupation free from liability, but about 75 per cent of the recorded cases have been in persons who by their occupation had to deal more or less directly with cereals, *e.g.* farmers, labourers, peasants, coachmen, grooms, millers, etc.

Treatment.—Various methods have been used, such as counter-irritation, mercury, arsenic, tuberculin, and other bacterial products, but iodide of potassium seems to be the only drug which should be relied upon; it seems, in fact, to have quite a specific action. The favourable results obtained by Thomassen have since been confirmed by Nocard, Ransom, M'Fadyean, and others. The dose for man is 30 to 60 grains daily (for cattle the dose is four times larger).

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Addison's Disease. *See* ADRENAL GLANDS.

Adenitis. *See* LYMPHATIC GLANDS.

Adenoma. *See* TUMOURS.

Adenoid Growths. *See* NOSE.

Adipocere.—This is the name given to a wax-like substance formed under certain conditions in the decomposition of dead bodies.

History.—Adipocere has been known for centuries, but attention was prominently drawn to it in 1786-87 when the *Cimetière des Innocents* at Paris was cleared out on account of the sickliness of the neighbourhood. In this graveyard great masses of bodies were found packed closely together, often with no earth or only decayed fragments of the coffins between them. Hundreds of these bodies had been converted almost completely into adipocere, in which condition they resisted decay. Even the bones in some cases had not escaped. Adipocere was found in the medullary cavities of the long bones, and in the smaller cavities of the spongy bones, the bones themselves in some cases being softened. These changes were described very fully by Fourcroy. Since then adipocere has been found not infrequently in bodies buried in damp soil, concealed in cess-pools, or in running water, or kept for anatomical purposes in cold damp cellars.

Description.— Adipocere, when dry, is a waxy substance resembling spermaceti in appearance; usually of a whitish colour, sometimes yellow or orange, or, especially on the surface, stained brown or black. Occasionally the surface has an iridescent appearance like a sheet of mica. Old specimens may be hard and friable. When more recent or when warm it is soft and ductile. Recent specimens differ also from old specimens in that they are more oily, they contain more water, and they often contain blood-vessels and other fragments of tissue. They differ also in chemical composition.

Chemical Composition.— Fourcroy considered adipocere an ammoniacal soap with excess of fat. Chevreul, as the result of analysis, concluded that adipocere was an ammoniacal soap containing a bitter substance, and an odoriferous principle with extraneous colouring matter, and small quantities of calcium and potassium; and this conclusion has been borne out by subsequent investigators. The principal acids present are palmitic, stearic, and oleic. Oleic acid is abundant in recent specimens, and may be absent from bodies that have been long buried. Calcium is found as calcium stearate or oleate chiefly in adipocere which has formed in hard water or in graves traversed by such.

Conditions of Formation.—Adipocere is formed most readily in bodies of children or of fat adults; in bodies buried in damp soils, especially those saturated by the drainage from privies or cess-pools; in water, and especially in running water. Cold favours its formation, and so, apparently, in some instances does heat.

Mode of Formation.—It is generally admitted that adipocere results, in part at least, from the saponification of the neutral fats of the body. Under the influence of putrefaction the fats split up into glycerine and fatty acids (including oleic acid). These acids unite with ammonia, derived from the decomposition of albumen, and so form ammoniacal soaps. In course of time much of the ammonia may be replaced by calcium derived from the water or soil in which the body lies. There has been much discussion as to the part played by the muscles and other albuminous tissues in the formation of adipocere. Fourcroy and others have maintained that these tissues simply supplied the ammonia necessary for the saponification of the fat. One difficulty in the way of acceptance of this view is that in many cases the amount of adipocere is far too great to be all derived from the fats originally present in the body. Accordingly, the view now generally held is that adipocere is derived not only from free fat, but from fat produced in the decomposition of albuminous material. That this is possible is supported by the analogies of fatty degeneration; of the ripening of cheese; and of the conversion into fat of albuminous food (Voit and Pettenkofer). Kratter supports this view by the result of microscopical examination of adipocere, and of neighbouring tissues.

Time required for Formation.—The time required for the production of adipocere has excited a great deal of interest and discussion, as it was at one time supposed that the amount of saponification present in a body might be important evidence of the time that had elapsed since death, and in several instances important legal decisions, especially as to survivorship, have turned upon this point. It appears, however, that the process goes on at no constant rate. Saponification may be as complete in a few months in one case, as after many years in another. Roughly speaking, it may be said that the formation of any notable amount of adipocere seldom occurs within six weeks in water, or three months in damp soil, while the complete conversion of a body into adipocere is a matter of years rather than months. In very favourable circumstances the time required may be considerably shortened, and very exceptional cases are quoted as having occurred, in India, in which an advanced degree of saponification was present within a few days of death.

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Adiposis Dolorosa. *See* OBESITY.

Adolescent Insanity.

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General Remarks.—The two great eras of brain development and brain decadence are periods of special danger to the mental functions, as they are indeed to all the nerve functions. In one case, a bad mental heredity will arrest and upset the normal and orderly development of mind, and we have idiocy or adolescent insanity resulting. In other cases, hereditary tendencies towards motor, sensory, or trophic weaknesses will result in epilepsy, infantile paralysis, or cleft palate. Careful and exact studies of brain development in all the multiform relations of the many and marvellous functions of that organ are yet wanting. The subject has lately received a great stimulus through Flechsig's observations on the development of the conduction paths in a child's brain in relation to the functional and organic requirements of the organism. In development, the evolution of sex and all that it implies, especially its mental relationships, are of supreme importance. In decadence, the extinction of reproductive energy affects mind profoundly, but not so greatly as its evolution. The mental changes of decadence have a closer relationship to diminished blood-supply and morbid vascular changes than to sexual abatement. The forms assumed by the psychoses and the neuroses of development and of decadence are in many important respects different, as might surely have been expected on physiological and psychological grounds. In both cases they are coloured by the special bodily and mental characteristics of each period of life. Speaking generally, the developmental insanities—not including the congenital mental arrestments of idiocy and congenital imbecility—are characterised by acuteness, exaltation, periodicity, and active sexual accompaniments; while those of decadence show more depression, neurasthenia, trophic abatement, amnesia, and psychopareses; their chief complications being the motor pareses.

Some Statistics.—A general idea of the liability of the different periods of life to nervous breakdown may be obtained by a glance at the mortality tables showing the causes of death from the different classes of disease at various periods of life. Looking at the numbers set forth in the Report of the Registrar-General for Scotland for 1895, we see that 55 per cent of the whole population is between the ages of 1 year and 25, both inclusive. More than one-half of the people of Scotland is, therefore, in the developmental stage as regards their brain and nervous functions. During this period, if the causes of deaths are inquired into, it is found that 35 per cent, or thereabouts, of the deaths have resulted from diseases of the nervous system. The brain is then evidently doing its work ill. That is in strong contrast to the vital statistics of the next period of life—that between 26 and 50 inclusive—which may be taken as the period of completed development, greatest bodily and mental strength, fullest reproductive power, and least liability to any of the failures of decay. The proportion of persons alive at this best period of life was 30 per cent. When the causes of death among this strong, active, responsible, sexual mass of humanity that is doing the chief part of the world's work is examined into, we find that only 12·2 per cent of the mortality results from nervous diseases. The brain is then clearly doing its work well. But when we examine into the same facts in regard to the next period—from 51 to 75—when life has begun to ebb and the energies to decay, though it cannot be said that the hard work and the strains of life are then so great as at the second period, we find that those who die, out of the 12 per cent which have remained alive, have fallen victims to the neuroses in the high proportion of 33 per cent. Clearly the brain is rapidly failing in its capacity through decline at this period. Curiously enough, and it is not easy to explain it, the sixth and seventh "ages"—the "lean and slippered pantaloon," and the "last scene of all," the second childishness and mere oblivion, "sans teeth, sans eyes, sans taste, sans everything"—from 76 to 100, constituting 3 per cent of the population, do not die from nervous failures in anything like the same proportion as the previous era, for only 14·8 per cent of the deaths then result from the neuroses. The general conclusions seem obvious. Nature finds it hard to build up a sound working brain with energising power enough and makes many failures in the attempt to do so, but when she succeeds and the organ reaches a fair standard of capacity at full development at 25 or so, it is not then apt to be overset till the natural period of its decadence is reached. When that occurs, a disproportionate number of men and women die of the neuroses. It is certainly not surprising that the brain and nerve-centres find development into full working strength a hard process, looking to their infinite complexity, and their inconceivable delicacy of structure. Nor is it wonderful, considering the frequent adverse circumstances of human upbringing and perhaps bad heredity with a not favourable environment, that the nervous centres are found so often of weak power and durability. And when we further consider the highest of all the brain's functions, that of mental action and its unthinkable relation to organisation, it is still less wonderful that failures and disturbances occur in the development and retrogression of the mental protoplasm.

Relation of the Abnormal to the Normal.—The insanities of development and decadence cannot properly be understood if they are not looked at in reference to the normal characteristics and activities, mental and bodily, of the early and delicate periods of life. The psychology of childhood, long neglected, is now a favourite, almost indeed a fashionable study, not only by the philosopher and the physiologist but by the teacher and the parent. It is a large and, as yet, only half-understood subject, and can only be referred to here. In relation to child-insanity—a very rare condition—the normal mental characters that are of most importance are the slight inhibitory power, the excitability, the vivid fancy, and the difficulty of most children in distinguishing between their fancies and facts, the great sensitiveness to some kinds of impressions and not to others, with the quick and often undue reactiveness, the keen amenability to sense stimuli, and the volatility of mind of children. These psychological characteristics, along with the tendency to that transient mental dissolution we call delirium, when the temperature becomes even slightly raised, taken together with the constant trophic changes which the mere growth and development of function of the brain imply, are the dominant points of the child's brain in action. But as the mental functions are not then fully evolved, neither do we then find systematised insanity common.

Then, what a change in the mental characteristics and activities of the brain do the advent and growth of sex produce! The intensification and special direction given to the affective faculties, the new organic cravings, the enlarged moral horizon, the new delights of the poetic and romantic in poetry and literature, all imply an evolution of function of the highest kind that had lain dormant before. The actual nutritional concomitants of sex, such as menstruation and the reflex influences from the sexual organs on the brain, are often disturbing to its equable working. The sudden appearance of intense altruism and the uprising of the religious instinct in its full strength change the whole "character." Above all, the hereditary instincts and the hereditary weakness that all the former generations have handed on to a man or woman, come first into view as regards the mental and moral nature between 15 and 25, and bring along with them great risks of mental disturbance. Little consideration is needed to show that all these changes are not sudden or rapid, but take years for their full development. A man or a woman's "character," power of inhibition and of endurance, and force of will, do not really develop at 15 but between 18 and 25, and that is found to be the most dangerous time for the onset of developmental or true adolescent insanity. That is, therefore, far more an adolescent than a pubescent psychosis. It is often assigned to puberty as a cause, but puberty is commonly years past before ever it appears.

THE INSANITIES OF CHILDHOOD, PUBERTY, AND ADOLESCENCE must all be regarded together if they are to be rightly considered. We may have *insanity in childhood*, but it is very rare. Brain instability seems to be represented in childhood by a liability to that short and acute psychosis we call delirium. The more neurotic the child the greater tendency for it to pass into a delirious condition, especially at night, on a very slight rise of temperature. Many a child with a strong neurotic heredity will become delirious at night with a temperature of 99°. There are many distinct varieties of delirium—the muttering, the screaming, that accompanied by elation and happiness, and that accompanied by fear and terror, that which almost entirely consists of vivid hallucinations, and that with intense motor explosions. Such delirium is not counted as technical insanity, however nearly allied it may be.

The form of child-insanity most common is that of mania, sometimes with great elation, extravagant conceptions, great restlessness, and rapidly changing fancies. A form less common is that where there is depression of mind, delusional fears, continuous weeping, and even suicidal impulses. Almost all child-insanity is commonly of short duration, but apt to recur. Frequently this form of insanity is not very well defined, but consists of the following symptoms mixed up with each other or in alternations, viz. quite unreasonable obstinacy and irritability, change of affection towards relatives, violence of a homicidal kind, shouting, swearing, smashing, causeless anger, loud wailing, fears, hallucinations of sight, covering of head and eyes, and, very occasionally, attempts at suicide. Not infrequently there occurs in certain neurotic children a condition of general mental explosiveness with sudden impulses in all directions. There can be no doubt that in most of those juvenile psychoses there is a mixture of some of the characters of delirium and epilepsy.

For the treatment of such affections I have found no drug so good in so many cases as the bromides given in full doses for the child's age, sometimes in very large doses indeed, and continued for many months after the acute symptoms have passed off. I remember giving a boy of eleven for an attack of maniacal violence and irritability 120 grains daily of the bromide of potassium at first, and he has taken 60 grains daily for the past two years. The effects were marked, both in subduing the excitement and irritability, and also in preventing altogether a recurrence of the acute mental symptoms. All the time he has grown, developed, and gained in weight.

Puberty and Adolescence.—It would be taking a narrow and unscientific view of the pathology of puberty and adolescence to forget that the psychoses of the period are merely one of a series of neuroses to which it is a subject. Chorea, somnambulism, megrim, epilepsy, hysteria, instinctive immorality, arrested body growth, and many skin and eye diseases are all prevalent and largely peculiar to this developmental era of life. The liability to acute rheumatism and phthisis pulmonalis is also, I believe, a common consequence of a neurotic heredity of this period. The typical adolescent insanity and pubescent insanity are almost identical in their characters. Adolescent insanity comes on most frequently between eighteen and twenty-five. It is, in fact, the last neurosis of development to appear. In 77 per cent of the cases the attack is maniacal in character, in the other 23 per cent it is melancholic or stuporous. The typical maniacal form commonly begins by a slight amount of depression, listlessness, incapacity to fix the attention, sleeplessness, and unsettledness. Soon there comes on a restless, elated, wayward, boastful, unconventional stage, often with erotic symptoms. The control of parents is resented, and a great delight is taken in shocking those connected with the patient. This state passes into one of acute mania, which lasts a few weeks or months in different cases. Persistent masturbation is apt to complicate this stage. Then all those acute and exalted symptoms abate and pass off, and a stage of confusion and sometimes of stupor, a sort of reaction after the excitement, comes on. After a few weeks the mental power improves until a state of apparent recovery takes place. But after a few weeks or in a month or two the patient passes through another such maniacal attack. Those relapses may take place many times before complete recovery takes place, or the patient sinks into secondary dementia through a stage of chronic mania. This tendency to short, sharp attacks, with intermissions of sanity, is more frequent in adolescent insanity than any other form of mental disease, except epileptic insanity and folie circulaire. Those apparent recoveries that do not last make it most disappointing to anxious parents, and to the doctors in attendance.

In addition to this periodic relapsing character there is another marked feature of the mania. The ideas, emotion, speech, and conduct are apt to be strongly tinctured by grotesque and exaggerated characteristics of normal adolescence. In addition to masturbation, hysterical symptoms in women, boastfulness, mock heroic motives, imitations of manly airs, obtrusive pugnaciousness, and a morbid sentimentality are very common.

In the melancholic adolescents there is not nearly the same tendency to relapse as in the maniacal cases. Frequently there are strong suicidal impulses that have to be guarded against. But for the most part the cases follow the lines of ordinary simple melancholia. One seldom sees the more serious examples of excited and restive melancholia at that age.

The cases of adolescent insanity which assume the form of stupor come next in frequency to the melancholic. Nearly all the typical cases of anergic stupor—the *primary dementia* and *dementia attonita* of many authors—are adolescents. This form of mental disease is a very interesting psychosis, most cases of trance and catalepsy being examples of it. There is often a sexual or masturbative element running through it. The vascular and trophic symptoms, lowness of temperature, a blue œdematous condition of the extremities, weak heart's action, tendency to chilblains, etc., point strongly to a neuro-vaso-motor defect.

There is a very rare form of adolescent insanity, which is the only one I admit to be a true primary dementia, in which there is no preliminary mania, or melancholia, or stupor, but a gradual enfeeblement of the mental powers until true terminal dementia is reached. I have only seen about six typical cases of this form. It is the only form that is hopeless from the beginning.

TREATMENT.—The treatment I have lately adopted for such cases is founded on physiological considerations. The normal completion of the period of adolescence is in both sexes accompanied by a considerable deposit of adipose tissue, by an overplus of strength and activity, and by a state of general good nourishment of the body. To attain to this normal condition of body should undoubtedly be our aim in treating all cases of mental disease at this period. It always seemed to me that there were two things that constantly worked the other way, and that I had to contend against in their treatment. These were the general brain excitability and the morbid strength, and often perversion, of the generative nisus, with the inhibitory power over it gone. The one tended to mania, sleeplessness, purposeless motor action, thinness, and exhaustion; the other to erotic trains of thought, sexual excitement, and masturbation. I found that inaction, reading, indoor life, and amusements increased the one, while novel-reading, solitariness, and long hours in bed aggravated the other, while animal food and alcoholic stimulants gave increased strength to both morbid tendencies. I therefore put my patients to active exercise in the open air for as many hours a day as possible, walking, digging in the garden, and wheeling barrows; I give them shower baths in the morning when the weather is suitable and they are strong enough, and I encourage active muscular exercise in every way. Athletic games of all sorts in the open air are certainly good as far as they go. I place great reliance on the diet. Milk in large quantity, and as often in the day as possible, bread, porridge, and broth are the staple articles of food for such patients here. My friend Dr. Keith, of this city, was the first to direct my attention to the advantage of a light farinaceous and milk diet in an allied class of cases, and my experience is strongly in favour of his views. The patients may have some fish, or fowl, or eggs, but in reality milk is the most important means of treatment. I seldom give such cases alcoholic stimulants. I give to all such patients who can take and assimilate it easily an emulsion of cod-liver oil, hypophosphite of lime, and pepsine, made and flavoured in such a way that it resembles cream or extract of malt. I find very few indeed who cannot take this. Beyond this, an occasional bitter tonic, with sometimes a chalybeate or some of the new compound syrups of the phosphates, are about all the medicines I give. The effect of this diet, regimen, and treatment is very marked in the majority of cases. No doubt during the first part of the attack patients may lose weight while the excitement is in its most acute stage, but they soon begin to gain weight, and my prognosis is always favourable when I find the patient beginning to gain weight within a reasonable time, say six months or so. I have had patients who, in spite of very sharp excitement indeed, and much sleeplessness, gained weight under this treatment. It seems to me that the process of fattening such a patient, and the conditions under which it takes place, are antagonistic to the disease and its results. I have known the stopping of the cod-liver oil to be followed at once by a loss or diminished gain in weight, and mental aggravation, and its resumption to be followed by improvement in both directions. If a young man or woman suffering under the insanity of adolescence is found to gain one or two pounds a week within the first three months, I look on him as pretty safe. It is common to gain a stone in a month.

I have now pursued this plan of treatment long enough to yield results that can be relied on, and I believe that more of my patients recover than before I adopted it. They recover sooner, and their recoveries are more reliable and permanent. Even in the case of those who sink into dementia I think they do so more quietly and with less of the element of chronic mania than under a flesh diet. It is, I think, certain that the habit of masturbation, which is so frequent and so deleterious in such cases, is less practised by patients on this diet and regimen, and, when practised, is less damaging to brain function, and takes less hold on them.

Prophylaxis.—Lastly, in connection with this subject, I would say a word about prophylaxis in children with a strong neurotic inheritance. My experience is that the children who have the most neurotic temperament and diathesis, and who show the greatest tendencies to instability of brain, are, as a rule, flesh-eaters, having a craving for animal food too often and in too great quantities. I have found also that a large proportion of the adolescent insane had been flesh-eaters, consuming and having a craving for much animal food. It is in such boys that the habit of masturbation is most apt to be acquired, and, when acquired, to produce such a fascination and a craving that it may ruin the bodily and mental powers. I have seen a change of diet to milk, fish, and farinaceous food produce a marked improvement in regard to the nervous irritability of such children. And in such children I thoroughly agree with Dr. Keith, who in Edinburgh for many years has preached an anti-flesh crusade in the bringing up of children up to eight or ten years of age. I believe that by a proper diet and regimen, along with other means, we can fight against and counteract inherited neurotic tendencies in many children, and tide them safely over the periods of puberty and adolescence.

Results of Treatment.—About 60 per cent recover; 30 per cent pass into secondary dementia; and 8½ per cent pass into other forms of insanity, chiefly chronic mania and delusional insanity. Insanity occurring at the adolescent period is therefore a curable disorder as compared with many other forms, though not so curable as puerperal insanity. When an adolescent becomes insane the patient is usually boyish or girlish in face and form, and when the recovery takes place the signs of completed physiological manhood and womanhood are apparent. The beard has grown, the form has expanded, the weight has increased, and the voice has fully changed. The mortality is very low—about 1·5 per cent.

SIGNIFICANCE OF ADOLESCENT INSANITY.—Looking to the gradual development of mind up to puberty, and the enormous and sudden leap that is then taken towards man's and woman's higher mental life, we must assume an almost complete cerebral apparatus lying ready to be brought into use, just as the centres of respiration are ready for their function at birth. Considering that the very highest mental, emotional, and moral qualities of a human being, with the subtile differentiations between the male and female types, only arise fully between eighteen and twenty-five in the average human being, it would be surprising indeed if the cortical working was not then liable to break down when we had hereditary weaknesses present. The most important, the most subtile, and the highest functions imply, of course, the most delicate brain mechanism and the greatest strain on the mechanism. This I believe to be the true theory of adolescents being so subject to attacks of insanity. It is truly a "critical" period of life to those with hereditary neurotic weaknesses. It is the statement of a fact when one says that all the cases of adolescent insanity have hereditary predisposition, mental, or, at all events, neurotic.

There cannot be the least doubt, I think, as to its relationship to the special mental characteristics of the period of life in which it occurs. It is the period of intense mental development in the female sex, tinctured at every point by her reproductive instincts, which are then acquiring solid form and physical potentiality. With reproduction comes like a flood the real knowledge and realisation of all the possibilities and all the mysteries of life. In the male sex all the combined ambitions and altruistic emotions of life then burst forth to consciousness. Up till then, in both sexes, sufficient had been the day for its pleasures and for its evils. A mild egoism had bounded the desires. No real intensity in life had appeared to carry everything before it. The philosophic meaning of the change is that the race is greater than the individual. We know that all this new mental life, all those intense emotions, all those overmastering ambitions, all those vague but profound longings, are represented, nay, that they arise out of brain energising. Not only do they do so, but also the ideal and the poetic of the period, and the very moral and volitional life, are dependent on brain working as much as the movements of the fingers. The undeveloped brain shows no overmastering intensities of feeling or volition, and has no impregnable moral resistances. When development proceeds normally every feeling and every mental capacity is expressed in a physical characteristic of expression, of form or of motion. The beauty of woman, and the strength and manliness of man, are the bodily equivalents and counterparts of the mental ideals proper to the sexes.

As it is during this period, from eighteen to twenty-five, that the mental "character" and the bodily "constitution" are fully formed and settled, it is no wonder that there are then risks to the mental functions of the brain to which hereditary weaknesses have been transmitted. If those weaknesses are mental the result is apt to be adolescent insanity, or one of that most varied group of mental and moral twists or lacks which may be described as adolescent torpor, and stupidity, and aimlessness; or adolescent perversion of the moral sense or power of control exhibited in incompatibility of temper, irritability, impulsiveness, dipsomania, immodesty, vice, shamelessness; or general lack of morality; or in changed emotion, such as utter selfishness, aversion to relatives, asocialness, and unnatural attachments. The bodily equivalents of such mental misdevelopments are menstrual irregularities, chlorosis, hysteria, neuralgia, unattractiveness of person, irregular or deficient growth of mammæ or beard, awkward body movements, and "bad" expressions of face.

There is, to my mind, a striking analogy between idiocy and adolescent dementia. With points of difference the great mental deficiencies are the same. The idiot has usually far more bodily defects, but that is because in idiocy the bad heredity was stronger and caused its effects during the formative and growth stage of life. I have seen cases where from traumatism, fevers, hydrocephalus, loss of the senses, or hereditary syphilis, children of from seven to twelve were arrested in their brain and mental development and left in states that could not be distinguished from congenital imbecility. There are a few cases of secondary dementia, resulting from attacks of adolescent mania, usually in the early years of adolescence, that become so complete as to resemble idiocy. Looking at the whole question of mental enfeeblement, from birth up to twenty-five, from the point of view of brain development, I am inclined to the conclusion that the secondary dementia of adolescent insanity is pathologically allied to genetous idiocy, as it certainly is etiologically through heredity. Much secondary dementia I thus look on as what might be called postponed idiocy. There are links that connect the one form of mental enfeeblement with the other which may occur in every year of life, from birth up to adolescence. Every form of enfeeblement, congenital or adolescent, may have maniacal attacks superadded to it. The percentage of deformed palates is nearly the same in idiocy and adolescent insanity, this frequently indicating, where it exists, a certain bad quality and non-development of brain.

To my mind the change in the brain cortex, through which cells that had, when normal, been the vehicle of high thought, keen emotion, vivid imagination, accurate memory, and powerful volition become, when pathological, unfit to manifest any of these qualities, is the most remarkable and mysterious change in Nature next to that from life to death, and of itself marks out adolescent mania as the greatest and most important of all the developmental neuroses.

Adolescent insanity has also this peculiarity, it occurs just before maturity. It is the last cortical developmental disease. Just when the organism is about to take on itself the burden of full reproductive capacity in addition to that of organic and mental life, it breaks down at the very highest point. Nature's law of the barrenness of a bad stock is thereby carried out, for dements, though not necessarily asexual or barren, are in any organised society non-reproductive.

The Lesser Mental and Moral Morbid Changes of Adolescence.—There are a series of lesser mental and moral changes and perversities that are liable to occur in adolescents of both sexes with hereditary weakness, short of mania or melancholia or technical insanity generally, that are frequently much more difficult to treat and often almost as distressing as the insanities, and which I believe to be due to the same hereditary and pathological causes, and to be of the same essential nature as insanity. They consist, in some cases, of stupidity and lethargy, so that the girl or lad ceases to care for or show intelligent curiosity in anything. Or they consist in an asocial development at this, normally, one of the most social of all ages. The youth ceases to mix with his friends, to play games, or to find pleasure in meeting his fellows. He shuts himself up, and his social instincts seem for the time paralysed. Or they take the form of a causeless aversion to father, mother, sister, or brother, intolerance of control by them, and utter disregard of their feelings. All this time he gets on well with strangers, who see nothing wrong. Or they take the form of "general incompatibility" of temper, so that the person gets on with no one, and is cantankerous and suspicious all round, losing situations, quarrelling with friends, and making enemies everywhere. Or they take the form of impracticability, with visionary scheming and want of common sense, of a mild paranoia. Or a frothy sentimental religionism develops itself, changing churches without reason, and taking always to new and striking modes of expressing their religious instincts. Or we have sudden immoralities contrary to the tenor of the former life. Or perverted sexual and reproductive trains of thought dominate the mind and affect the conduct. All these, being liable to occur as adolescent psychoses in hereditarily neurotic families, should certainly be looked on and treated from the medico-psychological rather than from the moral and disciplinary standpoints. Heredity and its effects on brain alone explain such divergencies from the normal, mental, and social types. They cause enormous misery in families, their real nature and origin are commonly misunderstood, and their kinship to mental disease is seldom thought of.

The morbid mental change, when it takes the form of simply diminution of the volitional power, so that irresolution, fickleness, want of power to follow any kind of continuous work, "morbid laziness," and lack of energy all round, are the chief characteristics, is particularly apt to be misunderstood.

I was once consulted about the case of a lady, who up to fourteen or fifteeen had been as ordinary children, but who since that age had been the despair of her teachers and the skeleton in the closet at home. While clever intellectually and not given to gross immorality of any sort, she had exhausted all the arts by which disobedience, lying, perversities of every kind, and outrageous unconventionalities of dress and conduct could break her parents' hearts. She seemed to be without affection, except towards animals, tramps, and oddities generally. She always professed sympathy with the bad, the low, and the unfortunate. Respectability was an unpardonable offence to her. She was amazingly ingenious in her ways of "shocking" her parents and their friends by word and action; yet she was well read; she would pass muster among strangers for a month at a time as a clever, interesting, and original girl. She was about to marry a robust but respectable clodhopper when I was consulted about her. She did so, and had one or two children, and became apparently a careful mother and a frugal farmer's wife; but she was always eccentric and asocial, and never professed any affection for her parents.

There are many such cases where the moral and mental "twist" seems to follow a "love affair" or disappointment, but in whom the "love affair," the disappointment, and the succeeding mental changes are, I believe, mere sequences in an adolescent mental neurosis.

I am quite convinced that losses of mental inhibition and morbid impulsiveness of all sorts, without intellectual defects, occur in neurotic adolescents as really pathological mental phenomena. I have elsewhere described adolescent dipsomania, and congenital absence of conscience and impulses towards crime. I think a recent criminal case in Scotland, that of the "Arran murderer," was an example of this. A weak, vain youth, with a notoriously bad heredity as regards mental taint, committed a murder under most extraordinary circumstances, without any real motive, and behaved in an insane manner afterwards, was convicted, but his sentence was afterwards respited as a result of a careful medical examination into his mental condition.

It would need much time to enter into the question of those most interesting phases of depression and transitory deep feeling that have been so common in the adolescent period of the lives of many men of genius. Literature owes much to them, and poetry too. What physician can read the lives and writings of Goethe, Carlyle, Cowper, or Thackeray without seeing that the world would never have had *Werther*, *Sartor Resartus*, or *Pendennis*, except their authors had passed through melancholic phases of existence at the time the reproductive nisus was rising into dominant strength over all the life and thoughts and feelings? Suicide was contemplated at the very time when those men were fullest of life. It did not then need disappointed love nor thwarted ambitions to make them "tired of life." Nor do we find such adolescent depression confined to men of poetic temperament. We know that men afterwards of calmest or most sceptical minds—political economists and philosophers like Stuart Mill and Hume—passed through melancholic periods from twenty to twenty-five, and both these men then seriously contemplated suicide. Any attempt to explain or to understand such "Stygian darkness, spectre-haunted," without taking into account the teachings of heredity and of the physiological psychology and pathology of development, would be entirely futile and misleading. Without going the length of Tolstoi, one could wish that biographers and writers of serious fiction more fully took into account the facts and the laws of physiology and heredity in doing their work.

DEFORMED PALATE AND BODILY ACCOMPANIMENTS.—The palate is deformed in 55 per cent of the adolescent insane—as compared with 61 per cent in idiots and congenital imbeciles, though the deformities are not usually so great. Commonly also the form and facial expression are neurotic, and the development of body and mind is retarded for its age. Frequently there are marked derangements of sensory neuroses, pains in the head are very common, explosiveness and undue impulsiveness are not uncommon, though I have met with many cases where there was unusual seriousness and thoughtfulness, with a want of the levity proper to their age in the young people who took attacks of adolescent insanity.

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Adrenal Glands.

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PHYSIOLOGY OF THE ADRENAL GLANDS

ANY knowledge or indeed serious research into the functions of the adrenal bodies dates from, and in fact was started by, Addison's discovery that morbid lesions in these bodies were associated with a train of symptoms. Addison himself supposed that the adrenal glands had a definite function, and this hypothesis soon received experimental proof from Brown-Séquard. By removing the adrenal bodies from dogs, cats, rabbits, and guinea-pigs, he came to the conclusions that these bodies were essential to life, and that death rapidly followed their removal, that the blood became poisonous, and that the adrenal bodies normally destroyed or modified a substance which had the property of transforming itself into pigment; when the adrenal glands were destroyed, this pigment collected in the blood, but the animals did not become pigmented, probably because death occurred too soon.

Soon after this, probably as a result of the predominance then attached to the influence of the nervous system and reflex action, the opinion became current that the disturbances following disease or experimental removal of the adrenal bodies were not due to the destruction or absence of the organs so much as to concomitant irritation and damage inflicted on the neighbouring sympathetic ganglia and plexuses. Observations were brought forward to show that careful removal of the adrenal bodies was not necessarily fatal; but in the light of our present knowledge it now appears probable that the glands were not completely removed, or that accessory adrenal bodies were left behind, and also that sufficient time was not allowed to elapse after the operation to say with confidence what the effects really were. This reaction of opinion lasted for a considerable period after Brown-Séquard's first published researches (1856), and the suprarenal bodies came to be regarded as being practically functionless.

Within recent years a vast quantity of experimental work has been performed on the adrenal bodies. Among the numerous observors, Tizzoni, Foa, and Pellacani, Marino-Zucco, Langlois and Abelous, and especially Oliver and Schäfer, are the most prominent.

The conclusion to be gathered from a survey of this recent work is that the adrenal bodies are necessary to life, and that their complete removal is followed by death, preceded by muscular prostration and other symptoms in some degree analogous to those seen in Addison's disease. In a few instances pigmentation has been produced by experimental removal of the adrenal glands, but it is quite exceptional. The function of the adrenal glands is manifestly not vicariously performed by any other organ when the glands are extirpated, otherwise death would not result.

Two views have been put forward as to the nature of this important function:—

(i.) That the adrenal glands are katabolic or excretory, that they get rid of, neutralise, or destroy effete blood pigment and toxins, and so prevent their accumulation in the body.

(ii.) That the organs are anabolic, and provide an internal secretion analogous to that of the thyroid body, which passes into the circulation and maintains the equilibrium that exists in health.

THE THEORY THAT THE ADRENAL GLANDS ARE EXCRETORY, or that they neutralise poisons circulating in the blood, rests mainly on the observations of Abelous and Langlois that the blood of animals dying from the effects of removal of the adrenal bodies is toxic, and that its effects on other animals more recently decapsulated is like that of curari. On the other hand, it is probable, as urged by Schäfer, that the blood of an animal dying slowly of any disease is toxic, and that its injection into an animal whose resistance has been diminished by a recent operation, would be followed by definite symptoms. The effects of injection of adrenal extract are extremely unlike those of curari, so that at any rate the adrenal bodies do not contain the poisonous bodies that they are supposed to neutralise or excrete. Again, the fact that the blood becomes toxic after removal of an organ can be explained as the result of abnormal metabolic changes that are then allowed to occur in other parts of the body, the restraining influence which is normally present being the internal secretion of the adrenal bodies.

There is no satisfactory proof that the adrenal bodies are excretory.

THE THEORY THAT THE ADRENAL GLANDS PROVIDE AN INTERNAL SECRETION which is necessary to health has been placed on a very firm basis by the experiments of Schäfer and Oliver. These observers found that the medulla of the adrenal glands yields an extract possessed of powerful physiological properties. Its effects are manifested on the heart, blood-vessels, and skeletal muscles. When injected into the circulation there is a marked rise of blood-pressure due to constriction of the arterioles. Oliver has shown by his arteriometer that the calibre of arteries in man diminishes under the influence of the extract. The constriction is not explained by stimulation of the vaso-motor centre, for it occurs after destruction of the central nervous system in frogs, or in a limb after section of its nerves; it is due to a direct action on the muscular tissue of the small arteries.

The effect passes off in a few minutes; when the extract is applied to the inflamed conjunctiva in man vaso-constriction is said to last fifteen minutes. The rapid disappearance is not due to excretion by the kidneys, for it is not affected in animals by clamping the renal arteries; probably the active principle becomes stowed away in the tissues, and especially the muscles of the body, as shown by the fact that the skeletal muscles show its effects for a longer time than the vascular system.

As a result of the high blood-pressure, the beat of the heart is enfeebled and slowed through the influence of the cardio-inhibitory centre in the medulla. If, however, the vagi are cut or their cardiac terminations paralysed by atropin the heart beats both more frequently and more powerfully than normally. The blood-pressure may then rise to four or five times its normal height.

The skeletal muscles are also markedly influenced by the extract; a single stimulation of their motor nerve gives rise to very prolonged contraction comparable to that produced by veratria. The muscle responds also to direct stimulation. These effects, as already pointed out, in no way correspond to those of curari.

When the extract is injected into an animal the knee-jerk disappears, and paralysis, which always appears first in the hind limbs, may follow a sufficient dose. The amount of the extract necessary to produce these effects is remarkably minute; about half a milligramme for each kilogramme of body weight produces a maximal effect. (Schäfer.)

A partial immunity has been observed by Swale Vincent after giving the extract.

The active principle of the extract is not obtainable from the cortex of the gland, but only from the medulla. It has been found that extracts from the glands in an advanced case of Addison's disease do not exert the specific physiological action, and, as is well known, the patients in this disease exhibit a great muscular debility and a low blood-pressure.

The nature of the active principle has not been definitely determined yet. It is not neurine, as was formerly suggested, or pyrocatechin. Moore suggests it is a derivative of pyridene, and quite recently Abel has separated the substance raising the blood-pressure from aqueous extracts of the glands in the form of a benzoate; to the active principle he assigns the formula $C_{17}H_{15}NO_4$.

The active principle is not destroyed by artificial gastric digestion *in vitro*, so that the administration of the extract by the mouth is physiologically correct from a theoretical point of view. There is also no doubt that it raises the blood-pressure of patients thought to have Addison's disease; this was very well shown in a patient under my care, whose blood-pressure went up very decidedly. The tracings were taken by Dr. O. F. F. Grünbaum, who, however, found that he could not raise his own blood-pressure by taking suprarenal extract by the mouth, as tested by Oliver's sphygmo-dynamometer. Swale Vincent also failed to produce any toxic effects in animals by giving suprarenal capsules and decoctions made from them by the mouth, and suggested that the active principle of the glands is not absorbed when taken into the stomach. Probably absorption from the gastric mucosa is slow, and the active principle as it is absorbed is stowed away on the muscles and does not influence the blood-pressure of healthy persons.

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Addison's Disease

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DEFINITION.—A constitutional disease characterised by an exaggeration of the normal pigmentation of the skin, extreme asthenia, and a tendency to syncope, nausea, and vomiting. During life no morbid lesion is discoverable, and after death alteration of the adrenal bodies is the chief or only change found.

History.—This disease was discovered by Dr. Thomas Addison, Physician to Guy's Hospital, while hunting for the cause underlying pernicious anæmia. His conclusions were first actually published as far back as 1849 in a communication to the now extinct South London Medical Society, and reported in the *London Medical Gazette* of that year; but it was not till five years later that his illustrated monograph on "the constitutional and local effects of disease of the suprarenal capsules" saw the light. But even then the recognition of this discovery was tardy, and it is possible that but for Sir Samuel Wilks' loyal energy the observations might have passed comparatively unnoticed in this country. In France, Brown-Séquard had at once been stimulated by Addison's discovery to investigate the physiology of the adrenal bodies. Trousseau, after a considerable interval, called the disease by Addison's name. The late Dr. Headlam Greenhow collected a large amount of valuable material in his Croonian lectures before the Royal College of Physicians in 1875. But from this time until after the discovery that myxœdema could be satisfactorily treated by thyroid extract, and the resulting physiological researches into the internal secretions of ductless glands, no special attention was paid to Addison's disease. In the last five years a good deal of interest has been manifested both in the pathogeny and in the treatment by adrenal extract of Addison's disease.

Frequency. Sex.—Addison's disease, though now thoroughly well known and recognised, is decidedly rare; it occurs more often in the male sex. In 183 cases regarded by Greenhow as unequivocal examples of the disease, 119 (or 65 per cent) were males, and 64 (or 34 per cent) were females; and in 49 cases recorded as occurring under sixteen years, 30 (or 61 per cent) were males, and 19 (or 39 per cent) were females. In Jaccoud's series of cases the proportion was much the same.

Age.—The average age at death is about thirty-one years; but instances at the extremes of life have been published, from seven days after birth, to eighty-one years of age. It is rare below puberty, but Dezirot has collected 48 cases under sixteen years of age. In connection with the observed fact that Addison's disease is rare in early life, it is of interest to note that in cases of tuberculosis of the other organs of the body, the adrenal glands appear to be remarkably immune to secondary infection in the early years of life.

Etiology.—Greenhow believed that the disease was disposed to by hard physical exercise, and by traumatism and local injuries to the back, and he connected with this theory the rarity of the disease among the well-to-do classes. It should, however, be borne in mind that not only are the well-to-do less numerous, but inasmuch as they are better fed they are less susceptible to tuberculosis, which is found to be the nature of the suprarenal lesion in about 80 per cent of the cases.

Very little is known as to its geographical distribution; Osler regards it as less rare in Europe than in America; very few cases have been described in the dark-skinned races of tropical countries, but this is probably due to the difficulty of recognising the disease clinically in the blacks.

It would naturally be expected that the disease would occur more often in families with a markedly tuberculous taint, but this is not as yet established. It is true that it has on two or three occasions been seen to occur in members of the same family (Andrewes, Schwab), and I have had under my own care a woman probably suffering from this disease, whose sister had died from it, and whose suprarenal capsules were examined after death.

Besides traumatism and blows on the back it is possible that hæmorrhages occurring into the medulla of the adrenal glands either at or after birth may so lower the resistance of the adrenal bodies as to render them more liable to tuberculous infection; while the organisation of blood-clot might lead to fibrosis and subsequent atrophy of the active portions of the glands, and so to Addison's disease of a non-tuberculous origin. In a number of cases tuberculous infection has been found to spread by continuity from tuberculous osteitis of the vertebræ. It may be associated with tuberculous disease elsewhere, especially in the lungs, and in children it is said to be frequently accompanied by tuberculous affections of the mesenteric glands. On the other hand, the only tuberculous foci in the body may be in the adrenal bodies.

Morbid Anatomy.—In his original memoir Addison described cases of tuberculous disease, of malignant disease of the adrenal bodies, one case of atrophy and fibrosis, and one case of a nodule of growth blocking the right adrenal vein with hæmorrhage into the gland. He then expressed his belief that the characteristic symptoms were due to interference with the function of the glands, however brought about, and not to the lesion being of any particular nature.

Sir Samuel Wilks, who early interested himself in Addison's discovery, paid special attention to the morbid anatomy of the disease, and became convinced that the lesion was always of the same kind, and that it was a primary inflammation comparable to hepatic cirrhosis. From his descriptions it is plain that the condition would now be regarded as tuberculous. This view, which may be called the Unity of Addison's disease, Dr. Addison appears later to have been inclined to adopt. Dr. Greenhow also supported this view, and no doubt the great majority of cases are of this nature; there is, however, a minority in which other lesions are present.

It will be convenient to describe seriatim the various conditions that have been met with in Addison's disease.

1. The Tuberculous Fibro-caseous Change.—This is found in from seventy-four to eighty per cent of the cases, and is in most instances bilateral; but contrary to what is sometimes said, a lesion of one adrenal body may give rise to classical symptoms of the disease, in fact this was so in one of Addison's orginal cases. On the other hand, caseous tubercle may be found in both glands in cases of death from other causes without any corresponding symptoms having been present.

The tuberculous process begins in the medulla by the appearance of discrete tubercles; these coalesce, and a mass of tuberculous granulation tissue, which is at first rather vascular and may appear somewhat pink on section, results. This stage is of short duration, and caseation soon supervenes; the medulla then appears firm, cheesy, and white. The tuberculous process is more marked as a rule near the hilum of the gland, and does not necessarily affect the whole of the medulla. After a time it spreads to the cortex, which, in like manner, becomes replaced by caseous material. The gland is then increased in size and weight and altered in shape. The tuberculous inflammation often extends to the adjacent tissues and leads to firm adhesions, which may involve the semilunar ganglia and solar plexus: in this way pain in the back may be set up.

The caseous material may become compressed by the surrounding cicatricial tissue, which contracts upon it and may, as time goes on, undergo infiltration with calcareous salts. On the other hand, softening may occur, and the glands may be mere sacs of caseous pus.

Tubercle bacilli have often been demonstrated, but by no means always. It is probable that they may die, and that in this way injection of the material into guinea-pigs, as performed by Delépine, may fail to lead to tuberculosis. It is, however, conceivable that some isolated cases of fibro-caseous change in the adrenal glands are due to syphilis and are gummatous.

2. Simple Atrophy has been recorded in some well-authenticated cases, and may be so extreme that the only trace found on most minute examination is a

slight yellowish pigmentation of the fat, suggesting in shadowy outline the form of the adrenal bodies; in other instances the glands have been described as being absent.

3. In some cases a process of ATROPHY WITH CHRONIC FIBROSIS is found without any evidence of tuberculous activity. The late Dr. Haddon compared this lesion to that seen in the thyroid gland in myxœdema.

4. In MALIGNANT DISEASE involving the adrenal bodies the typical clinical picture of Addison's disease is generally not presented; but some or all of the special symptoms, pigmentation, asthenia, gastric disturbance, are occasionally seen, and are no doubt due, in part at least, to the adrenal bodies being involved. When secondary malignant growths of the adrenal bodies are present the primary growths may give rise to symptoms which mask or throw into the shade those due to the affection of the adrenal bodies. Sometimes symptoms like those of Addison's disease supervene in the course of adrenal malignant disease, just as pernicious anæmia may in carcinoma of the stomach. But in such a case the patient is really suffering from two morbid entities, malignant disease and Addison's disease. It is not to be wondered at, therefore, that these cases do not run the ordinary chronic course of Addison's disease.

Sometimes both the glands are extensively infiltrated with secondary growth, and yet no symptoms have been set up; occasionally in tuberculosis the same thing is seen. Such cases are at first sight somewhat difficult to explain, but it is probable that the patients have died of the primary disease before secondary symptoms due to the secondary adrenal changes have had time to develop.

Symptoms suggesting Addison's disease have more often been noticed as a result of secondary growths in the adrenal bodies than in primary malignant disease of the organs; probably because primary growths are not only rarer, but rather less likely to involve both glands. Still, some of the characteristic symptoms have been observed in primary malignant disease of the adrenal bodies.

5. In a very few cases symptoms thought to correspond to those of Addison's disease have been found associated with the presence of *extravasation of blood* into the substance of the organs.

6. In 12 per cent of the cases of Addison's disease the adrenal bodies have been found to be *normal*. In some of these the parts in the neighbourhood have been invaded by lymph-adenomatous or other growth, surrounded by adhesions or exposed to pressure. Under such conditions the nerve-fibres of the solar plexus and the semilunar ganglia may be irritated or destroyed, while the veins and efferent lymphatics of the adrenal bodies may also be compressed and the passage of fluid through them interfered with.

Those cases resembling Addison's disease, where after death the adrenal bodies and all the neighbouring structures are described as quite normal, are difficult to explain. It may be suggested that though anatomically normal the glands were functionally inadequate, or that the diagnosis of Addison's disease was open to doubt.

THE CONDITION OF THE SYMPATHETIC NERVES AND GANGLIA in the immediate neighbourhood of the adrenal bodies in cases of Addison's disease has naturally attracted much attention, since it has been widely held that the symptoms of the disease were due to irritation of, and subsequently the degeneration of, the semilunar ganglia and branches of solar plexus, and thus only indirectly to the lesions in the adrenal glands. To the naked eye the semilunar ganglia and solar plexus may, on careful dissection, be found to be enclosed in dense fibrous adhesions, and the nerves running into the fibrous mass to be thickened. On the other hand, as in cases of simple atrophy of the adrenal glands, the nerve ganglia and fibres may appear absolutely healthy.

The results of microscopic examination on this point are not harmonious; in a minority the sympathetic is found to be normal, while in the majority changes have been described. But it is important to remember that many of these changes correspond with what is normally seen in the semilunar ganglia of adult human beings. A microscopic examination should always be made at the same time of the semilunar ganglia of another person of the same age, and the sections of the two compared. It may be safely said that the conditions of the semilunar ganglia and adjacent sympathetic nerves in Addison's disease are so inconstant that the changes described in them cannot be regarded as causal factors in the manifestation of the disease.

OTHER ANATOMICAL CHANGES.—Besides primary tuberculosis elsewhere in the body, especially in the lungs, the mesenteric and retro-peritoneal glands may be tuberculous.

The lymphoid follicles at the pyloric end of the stomach may be unduly prominent, and occasionally, by breaking down, may give rise to small ulcers in the mucosa. The lymphoid follicles in the intestine may also be exaggerated.

The spleen is sometimes enlarged and softened, suggesting some toxic condition; this enlargement, however, is not constant, and hence it is probably the result of a terminal infection, such as so commonly proves fatal to patients with chronic disease.

The thymus gland is sometimes persistent and enlarged; this may be a mere coincidence, but it is interesting to note that it is enlarged in exophthalmic goitre, sometimes in myxœdema, cretinism and acromegaly—diseases of other "ductless glands."

Pigmentation of the peritoneum and even of the intestinal mucosa has been met with in exceptional instances, but is probably due to cured tuberculous inflammation that has left no other evidence of its existence, and is not analogous with the pigmentation of other parts of the body.

A few isolated reports of a softened condition of the brain and of degeneration in the spinal cord have been published, but no importance can be attached to them until more ample observations are forthcoming; they may have been merely accidental.

The heart is flabby, pale, and may be atrophied. Thus in a woman, aged twenty-eight, who died of the disease, it weighed only 4¾ ounces.

Pathology.—The relation between the morbid lesions in and about the adrenal bodies on the one hand, and the manifestations of the disease on the other, is a subject of great theoretical interest. Space does not allow of a full discussion of the problems, and I may perhaps be allowed to refer the reader for this to the article in Allbutt's *System of Medicine*, vol. iv.

The theories as to the method by which the symptoms are produced are:—

1. *The Nervous Theory.*—This is based on post-mortem dissection, showing that inflammation spreads from the adrenal bodies to the adjacent semilunar ganglia and sympathetic. The important element is the lesion of the sympathetic; this is usually secondary to tubercle of the adrenal bodies, but it may be entirely independent of any change in them. The changes are first irritative, and thus give rise to vomiting and pigmentation, and later become atrophic and degenerative, and are then correlated with great debility and collapse.

This theory explains satisfactorily cases where the adrenal glands are found to be healthy and where the sympathetic is involved in growth or inflammatory adhesions.

It does not, however, explain cases where the sympathetic has been carefully examined and found to be normal. According to Arnaud and Alexais, however, the important lesion is an affection of the ganglia embedded in the capsule of the adrenal bodies; this lesion may be well marked when the semilunar ganglia and sympathetic are healthy, and thus the nervous theory can be upheld even in the absence of any change in the abdominal sympathetic and semilunar ganglia. But the comparative rarity of ganglion cells in the capsule of the adrenal bodies renders this hypothesis somewhat unstable, and in any case it will not hold for cases of Addison's disease due to simple atrophy of the adrenal bodies. The nervous theory will therefore not explain all cases of Addison's disease, and cannot be considered as exclusive.

2. *Theory of Adrenal Inadequacy.*—This postulates that Addison's disease is analogous to myxœdema, and that it is due to an absence of the internal secretion of the adrenal glands.

The absence of the internal secretion might theoretically lead either to a condition of general apathy and atony, or, upsetting the balance of metabolic processes in the economy, to a condition of toxæmia. Possibly both factors may play a part: at any rate, in the present state of our knowledge it is impossible to be more precise.

The evidence in favour of this theory is (*a*) that the adrenal bodies in health contain in their medulla an active principle which raises blood-pressure and exerts a marked influence on muscular tissue; (*b*) that this active principle has been shown by Oliver and Schäfer to be absent in the adrenal bodies from a case of Addison's disease; (*c*) that muscular weakness, low blood-pressure and cardiac weakness are marked features of Addison's disease; and (*d*) that these symptoms may be relieved by the administration of adrenal extract.

Against this theory it might be urged that in some cases the adrenal glands are healthy, and that the sympathetic is involved in growth. In reply to this it might be said that the growth may very probably compress the efferent vessels from the glands, and so prevent the secretion getting into the circulation. Another objection is that, as already pointed out, the adrenal glands may be extensively destroyed by secondary growths without any symptoms of Addison's disease; it is possible that in these cases accessory adrenal glands were present, and able to provide sufficient internal secretion, and that compensation was thus brought about, or that death from the primary growth supervened before the symptoms due to the secondary destruction of the adrenal bodies had time to develop.

Of the two theories, that of adrenal inadequacy is the more probable, but there is one strong objection, viz. that whereas the administration of adrenal extract does good in some cases, it does not have much or any effect in a large number. This fact points to the probability that neither of these two theories is exclusively right or wrong, and that there is a third conceivable explanation of the relationship between the symptoms of Addison's disease in its morbid anatomy.

3. *Theory of the Duality of Addison's disease.*—This is a compromise, and has the advantage that it will satisfactorily explain, as far as is known at present, all cases of Addison's disease. It has been recently advocated by Byrom Bramwell and Boinet.

Some cases of Addison's disease are due to adrenal inadequacy alone; these are relieved by the administration of the extract, and are associated with atrophy of the adrenal glands. In the other and the larger number of cases, which are but little benefited by taking adrenal extract, two factors are at work—(*a*) adrenal inadequacy, (*b*) irritation of the abdominal sympathetic.

If this theory is correct, the effect of adrenal extract would be of considerable value in prognosis.

Onset and Course.—The disease begins insidiously, the patient having been in indifferent health for some considerable time before coming under observation. In rare instances the symptoms come on suddenly, and the disease runs a rapid course. This sudden onset may be caused by a shock or sudden depressing circumstances; in these cases there may be no pigmentation. The gastro-intestinal symptoms and debility as a rule appear first, and after some interval pigmentation follows; occasionally this order is reversed. The disease varies very much in its course, it often remains stationary for a time; its progress is distinctly irregular. There are exacerbations with periods of quiescence between, but after each paroxysm the patient is left a little worse than before the attack, though his symptoms are less marked in the remission than during the preceding paroxysm.

Symptoms.—The most important symptoms are referable to the muscular, vascular, gastro-intestinal and cutaneous systems; it will be convenient to consider them seriatim.

Asthenia is the most important constitutional feature; it occurs very early in the course of the disease and becomes progressively worse. The patient is always tired and languid, and eventually becomes utterly incapable of muscular exertion. The loss of power is out of all proportion to the state of nutrition. Langlois indeed lays stress on the total loss of sustained muscular effort which distinguishes Addison's disease from phthisis and other debilitating diseases, and suggests that when there is doubt as to the diagnosis Mosso's ergograph should be employed.

The action of the heart is feeble and the pulse very small, soft, compressible, and sometimes imperceptible. Syncope is likely to take place, and cardiac debility is further shown by palpitation, sighing, yawning, and the difficulty of feeling the cardiac impulse against the chest or appreciating the pulse at the wrist. Usually the pulse-rate is about the normal, but it may be slow, in rare instances, to a marked degree. The tension as estimated by the sphygmo-dynamometer is very low. Dyspnœa on exertion, probably cardiac in origin, may be a prominent feature.

Anæmia is not a marked symptom; secondary anæmia occurs, and the blood count is, according to Osler, usually 50 to 60 per cent of the normal, but more marked anæmia is occasionally seen. Leucocytosis, if present, is independent of the disease. The presence of black pigment granules in the leucocytes has been described, but requires further confirmation.

The patients are generally thin, but very rarely become emaciated, the muscles waste, but the subcutaneous fat remains. In exceptional instances they may lose flesh rapidly and to a marked degree. The temperature is as a rule subnormal, and the extremities chilly; fever has, however, been known to supervene before death. Cold is badly borne by the subjects of this disease.

The gastro-intestinal symptoms are of two kinds, those due to irritation, such as vomiting and diarrhœa, and those of debility and loss of tone—anorexia and constipation. *Vomiting, nausea, and retching* are frequently prominent features and may persist throughout the course of the disease, and be so irrepressible as to lead to death from exhaustion. Flatulence may be troublesome. Diarrhœa is comparatively infrequent, but it may be so severe as to bring the patient to the verge of death, and is more often seen late in the disease. The appetite is easily impaired, and may later in the course of the illness be succeeded by loathing of food. The bowels are usually confined.

Pigmentation is of all the symptoms the most likely to suggest Addison's disease in a case of obscure debility, and must therefore be regarded as an objective symptom of great importance. It generally appears some time after the constitutional symptoms of asthenia and vomiting, and in cases where the disease runs a rapid course it may never develop; these are cases of Addison's disease without bronzing of the skin, and are therefore less easy to diagnose. Conversely, though but rarely, the bronzing may exist for some years before there are any constitutional symptoms of the disease.

The subject of pigmentation may be considered under two heads—(*a*) as it affects the skin; and (*b*) the mucous membrane.

(*a*) The cutaneous pigmentation is an exaggeration of the normal, the pigment—melanin—is the same as that normally found in the skin, and pathologically in melanotic sarcoma. It is manufactured by the cells of the stratum Malpighii, and is not like the pigment in malarial melanæmia or in cases of pigmentary diabetes directly formed from hæmoglobin. Melanin is free from iron and contains sulphur. Though not derived directly from the blood pigment, its production in the skin varies with the vascularity of the underlying dermis. The material from which the melanin is manufactured would appear to be conveyed to the stratum Malpighii by "carrier cells" from the vessels of the dermis. The pigment is situated in the cells of the stratum Malpighii, and little or none can be detected in the superficial layers of the stratum corneum. The cutaneous pigmentation is distributed much on the same lines as in health, of which it is merely an exaggeration. It is found on the exposed parts of the face, neck, and backs of the hands. The palms of the hands and the nails are not affected or the parts as the face covered by hair; the evolution of the pigment is in some degree dependent on the stimulation of the actinic rays, and the absence or late development of bronzing may, perhaps, thus be explained in certain instances. On the covered parts of the body, the situations where pigment is especially found normally, or where there is friction and irritation, are found to be bronzed. Thus the areolæ, the backs of the forearms, the anterior folds of the axillæ, the genitals, the linea alba, the waist, the knees where garters are worn, the shoulders under braces, and the spines of the vertebræ become pigmented. Around scars the skin becomes darker, pock marks may thus become accentuated, and the skin that has been irritated by the local application of a blister may be permanently mapped out. The hair also becomes much darker. Small black spots like minute moles may develop, and are often considered of special diagnostic value.

The pigmentation of the skin is usually not uniform, being as already pointed out more marked in certain positions. Its degree varies considerably, the skin may be of a dirty hue, brown and imitating the effects of prolonged exposure to the sun, and in rare instances of a dark olive colour, making the patient look like one of the dark-skinned races. In its irregularity the bronzing of Addison's disease may resemble that of pregnant women. Occasionally the bronzing of true Addison's disease is associated with leucoderma. In connection with the fact that the pigmentation may sometimes resemble that seen in dark-skinned races, it is interesting to note that a peculiar odour is sometimes present, and that it has been described as resembling that emanating from negroes.

(*b*) Pigmentation of mucous surfaces is much less constant, and is probably more directly dependent on irritation for its evolution. Generally it is regarded as of considerable diagnostic importance. Personally, since it is often absent and has been known to occur under other conditions, I consider that rather too much stress has been laid upon it.

When present it is situated in the corium, and not in the stratum mucosum (Dixon Mann). It is seen on the lips along the lines of mutual contact; inky stains may occur on the side of the tongue, on the buccal mucous membrane, or on the gums, and may be, but not constantly, found to be associated with the irritation of carious teeth. It should be noted that pigmentation of the mouth is commonly seen in Lascars, in blue-gummed negroes, and very exceptionally in healthy persons, while it has occasionally been observed in other abdominal diseases, such as gastric ulcer, carcinoma, cirrhosis of the liver, and cholangitis.

As a rule the conjunctiva is anæmic, and contrasts with the bronzed skin of the face, but occasionally the conjunctiva shows pigmentation. It is said that the vagina may show pigmentation.

Nervous symptoms, headache and vertigo, the latter probably being allied to faintness; are not uncommon. Pain in the lumbar region is frequently complained of, and may well be connected with the dragging of adhesions around the adrenal bodies.

Pain is also often felt in the epigastrium and hypochondriac regions as well as in the limbs and joints.

Vision and hearing may be impaired, and there is often great mental apathy, but the mind remains clear unless coma or a low form of delirium supervene. When unconsciousness comes on there may be muscular twitchings or convulsions. Convulsions are more frequent in those exceptional cases that occur in early life.

The urine does not present any constant or characteristic features. Albumin and sugar are absent. There is no satisfactory evidence that it is more toxic than in health, or that any abnormal pigment is excreted in it. In fact the urinary pigments are rather diminished in amount than otherwise. Indican is sometimes present, but is not of any special importance. The specific gravity is low and the amount of urea diminished, but this is probably related to the diminished intake of food. In women, menstruation is as a rule irregular or suppressed.

THE DURATION of the disease is extremely variable, it has been known to extend over more than ten years, while on the other hand there are acute cases that rapidly prove fatal. In cases where convulsions or collapse suddenly occur and terminate life without constitutional symptoms having been previously noticed, examination of the adrenal glands shows that the lesion is of considerable standing, and that the disease has apparently been latent. The acute development of symptoms has in some instances followed on a severe shock or depressing circumstances, such as a violent purge.

Probably the average duration is about 2-3 years.

Death may occur in various ways. There is a marked tendency to syncope, which may prove fatal unexpectedly, and, sometimes from the slight degree of the symptoms, somewhat prematurely. In some cases the end comes gradually and quietly in a state of great debility, or may be precipitated by severe vomiting or diarrhœa. In other cases death is preceded by delirium, coma, or convulsions.

THE DIAGNOSIS of Addison's disease is in well-marked examples a matter of no difficulty, but in a great many instances it can only be arrived at by a process of excluding any other conditions that can produce the same symptoms. It may be convenient to consider the diagnosis under the two heads of (i.) cases with, and (ii.) cases without pigmentation.

(i.) Pigmentation with debility and vomiting may occur in *other abdominal diseases*, and sometimes when there are growths or lesions in the neighbourhood of the adrenal bodies, the condition if not Addison's disease is, at any rate, often indistinguishable from it. Such cases are those of lymphadenomatous formations in the upper part of the abdomen. In chronic peritonitis of tuberculous or malignant origin, in a few instances of hepatic cirrhosis, in pancreatic disease, in uterine disease and other conditions the face may show considerable pigmentation, but the remainder of the body is not affected in the same way.

There is a rare disease, acanthosis nigricans, nearly always associated with malignant disease inside the abdomen, where in addition to being pigmented the skin becomes rough and warty, and is thus differentiated from Addison's disease.

Diabetes may be accompanied by pigmentation—bronzed diabetes—but here an examination of the urine should prevent any mistake. Jaundice should hardly give rise to any difficulty, though "urobilin jaundice" is more likely to resemble the bronzing of Addison's disease.

Pregnancy, by giving rise to pigmentation, amenorrhœa, and vomiting, may imitate the disease, but the examination of the mammæ and abdomen should never be omitted when this fallacy is possible. In some cases of granular kidney the face is distinctly pigmented.

In phthisis also the face may be so pigmented as to suggest some secondary infection of the adrenal bodies.

In advanced *malarial cachexia* the skin is discoloured from melanæmia, but the history should prevent any difficulty arising. A general pigmentation of the skin has been known to occur in large melanotic growths of the liver. In exophthalmic goitre, rheumatoid arthritis, and cases where arsenic has been taken for a long time, pigmentation of the skin may be very noticeable. In generalised sclerodermia the skin may be darkened, but it is at the same time hard, and thus differs from what is seen in Addison's disease. Nitrate of silver may also produce a permanent discoloration of the skin—argyria—which, however, is very rarely seen, and is not accompanied by the constitutional symptoms of the disease. Long-continued irritation of the skin by pediculi and abstinence from washing may lead to great alteration of the colour of the skin; this condition ("*vagabond's disease*"), if combined with insufficient food, may suggest Addison's disease until simple treatment with soap and food alters the aspect of the case. In syphilis the skin may be darkened, as is also seen in pellagra. In the dark races debility due to beriberi or other causes must not be forgotten, while the influence of exposure to the sun, to tar, coal, and great heat on the skin must be borne in mind.

(ii.) The diagnosis of Addison's disease without bronzing is very difficult, and depends on the presence of marked constitutional symptoms without any other adequate cause. It must be distinguished from the *severe forms of anæmia*. In pernicious anæmia the examination of the blood should be sufficient, while in splenic anæmia the enlargement of the spleen would at once raise grave doubts as to the diagnosis of Addison's disease. In cases of latent carcinoma of the stomach, examination of the vomited matter might be of value.

THE PROGNOSIS of cases that are sufficiently well marked to be recognised is very bad indeed, and death sooner or later occurs, but the interval is very variable. In some cases arrest of the symptoms may occur, and this may be due to the administration of adrenal extract. Though it is probable that the disease may become latent, cases reported as being cured always raise the question of the possibility of an error in diagnosis, since a positive diagnosis is often far from easy.

If the disease is in some cases due to adrenal inadequacy, and in other cases due to that factor and irritation of the adjacent sympathetic combined, the administration of adrenal extract, which very probably relieves patients in the first category but is powerless for good in the more complicated cases, should be of value in prognosis, since the prognosis would be less gloomy in the simple but, unfortunately, rarer cases of simple atrophy of the adrenal bodies giving rise to glandular inadequacy.

THE TREATMENT may be considered under two heads: (1) the general lines of symptomatic treatment; and (2) the special treatment by the administration of adrenal gland substance—opotherapy.

(1) The general lines of treatment consist in preserving the patient's strength, guarding against any depressing factors, and the amelioration of symptoms as they arise.

Food of a nutritious and easily digestible character should be given, and stimulants such as champagne or whisky are generally required. Cod-liver oil in an emulsion and malt extract should be given unless it disturbs digestion or sets up looseness of the bowels. As tonics, strychnine and arsenic should also be given, care being taken to prevent any gastro-enteritis from the latter. If asthenia is at all marked, the patient should remain entirely in bed and should not be allowed to raise his head, as there is great danger of sudden and fatal syncope.

The patient will be unable in nearly all instances to perform his ordinary work, but he must be protected from worry and undue excitement, and must not be allowed to over-exert himself. Exposure to cold, which is borne badly by the subjects of this disease, must be avoided.

Constipation should be combated by mild remedies, for severe and even fatal consequences have followed vigorous purgatives. Diarrhœa should be met by bismuth and opium, while vomiting requires most careful feeding, ice, liquid, predigested food in small quantities at frequent intervals and effervescing drinks, while bismuth, opium, and oxalate of cerium should be tried.

(2) TREATMENT BY ADRENAL SUBSTANCE.—This has been given in various ways with the view of supplying the internal secretion of the glands. It has been given hypodermically or by the mouth, in the form of raw adrenal glands, as a tincture, and usually in compressed tabloids containing the extract of the glands of sheep.

Schäfer and Oliver found that the active principle is not destroyed by artificial gastric digestion *in vitro*, and hence the administration by the mouth instead of subcutaneous injection appears theoretically correct, but Swale Vincent's observations, already referred to, throw some doubt on the efficacy of adrenal substance given by the mouth. There is no doubt, however, that some cases of Addison's disease do improve markedly after the administration of the extract by the mouth, and the possibility arises that by a rise of blood-pressure after adrenal feeding, patients with the disease might be distinguished from healthy persons.

When the extract is given by the mouth, as is ordinarily done, 15 grains of the gland is a convenient quantity to begin with; this can be increased gradually. As much as two drachms of the gland substance have been taken daily without any bad effects; but the administration should be conducted with caution. Markedly depressant effects accompanied by febrile disturbance have been noted in some cases, and it is advisable that the treatment should only be followed out under circumstances admitting of careful observations of the case.

Examination of published cases shows that the results are variable, sometimes great improvement or even cure is recorded, sometimes temporary improvement with relapse in spite of continued treatment, and unfortunately in about half the cases no improvement at all. In connection with the temporary amelioration observed in some instances it is possible that this was due to the effects of suggestion, or that it was merely a phase in the course of the disease and quite unconnected with the administration of the active principle of the glands. The disappointing and inconstant results of adrenal extract certainly support Dr. Bramwell's suggestion that while some cases — those that improve on this form of treatment — may be due to adrenal inadequacy, the remainder are due to an additional lesion of the neighbouring sympathetic.

The lesion, as far as can be seen, is not adapted to surgical interference, though it is true that one case recovered after removal of a tuberculous adrenal body. The operation was performed for removal of what were supposed to be enlarged lymphatic glands.

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Other Diseases and Morbid Conditions of the Adrenal Glands.

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Fatty Change.—In adult life the cells of the cortex very commonly contain globules of fat, but in children there is little or no fat normally. In toxic conditions and in marasmus, Attlee has found that the adrenal bodies of infants are more constantly affected by fatty degeneration than the liver. His experiments on animals showed that toxic and septic conditions and starvation led to the same result.

Hæmorrhage into the adrenal glands may be met with in a variety of conditions.

(1) Traumatic, and due to some severe injury, such as fractured spine; in such cases there may be extravasation of blood around the adrenal bodies and into their substance, usually into the medulla. In difficult labours the infants not uncommonly show extravasation into the medulla. In 105 still-born children Dr. H. Spenser found hæmorrhage into the adrenal bodies in twenty-three cases, and in twenty-five children dying before the fifth day of life one showed hæmorrhage.

(2) In asphyxia, hæmorrhages occur into the lax tissue of the medulla; it may also occur in cases of chronic venous congestion. In these cases the cause of the hæmorrhage is partly mechanical, but also perhaps partly toxic.

(3) In toxic conditions. Experimentally the injection of diphtheritic and other toxins has this effect. In man hæmorrhage may occur into the adrenal bodies in purpuric conditions. Cases in which hæmorrhage into the adrenal glands occurred in unvaccinated infants and suggested the possibility of variola have been recorded by Andrewes, and Garrod and Drysdale. Occasionally in other toxæmic conditions hæmorrhages are found.

(4) In septicæmia and pyæmia. In septic and toxic conditions the adrenal bodies are swollen and their cells in a condition of cloudy swelling. The organs are softened, and on slight manipulation the medulla may give way so that the organ appears to contain a cavity. Embolism in pyæmia may give rise to small hæmorrhages, and both it and in septicæmia the organs are sodden, engorged, and may show extravasations.

Suppuration may occur in pyæmia, as already stated, but the abscesses are small and of no clinical significance. Abscess in the suprarenal bodies without disease elsewhere is one of the rarest lesions. Caseous tubercles may soften down and imitate an ordinary abscess.

Syphilis.—In congenital syphilis hæmorrhages sometimes occur into the adrenal bodies, and may be explained as toxic in origin. But as a result both of congenital and acquired syphilis, gummata, usually of small dimensions, may occur, but they are rare.

A calcified gumma has been described in a girl two months old with extensive congenital syphilitic lesions. Fibrosis and atrophy may exceptionally occur in the subjects of the acquired disease.

Tubercle.—In generalised tuberculosis miliary tubercles may be met with. Chronic tubercle always begins in the medulla, and not uncommonly occurs without any clinical manifestations of Addison's disease. In 157 cases of tuberculous disease of various parts of the body this was observed in twenty cases; it further appeared from an analysis of these observations that the adrenal bodies were less susceptible in early life than later on.

LARDACEOUS DISEASE.—The adrenal bodies are not infrequently affected; thus in twenty-one cases of lardaceous disease the adrenal glands were involved in nine. It is seen in the vessels of the cortex. It has no clinical significance.

SIMPLE TUMOURS OF THE ADRENAL GLANDS.—Adenomata, composed of encapsuled collections of adrenal cells, are the only simple tumours that are met with under ordinary circumstances. Several forms of adenoma are met with.

(1) Small encapsuled nodules projecting from the cortex; they are frequently multiple. On section they are yellowish-white, and must not be mistaken, as they sometimes are, for discrete caseous tubercles. These small adenomata are indistinguishable from accessory adrenal bodies embedded in the cortex of the main gland; possibly this is what they really are. They are also superficially like the irregularities not infrequently seen on the outside of adrenal bodies in advanced life, but differ from them in having a fibrous capsule.

(2) Larger single adenomata arising from the cortex and reaching on an average the size of a walnut (struma lipomatosa suprarenalis, Virchow). Occasionally they are bilateral. They are composed of fatty adrenal cells and are derived from the cortex. The columns of cells are separated from each other by delicate connective tissue trabeculæ, in which blood-vessels are found. As the result of increase in the amount of this fibrous stroma, the appearance of a fibro-adenoma may be produced, and in these rare cases hyaline degeneration of the fibrous tissue may occur. The adrenal cells, which are ordinarily fatty, may soften down, and a pseudo-cyst thus be formed. Hæmorrhage from the vessels sometimes occurs, and may give rise to a malignant-looking tumour; it is highly probable that some cases formerly described as sarcoma, without any secondary growth or invasion of adjacent organs, were hæmorrhagic adenomata. Letulle has specially drawn attention to diffuse hæmorrhagic adenomata arising from the cortex, which are in reality innocent tumours, but are often erroneously regarded as sarcomata. These large adenomata are the only ones of any clinical significance.

(3) Letulle has described multiple pigmented adenomata in the cortex; the cells are not fatty. He observed three cases, all in patients with phthisis.

(4) Adenomata arising from the medulla of the adrenal body are rare. They are vascular; the capillaries running between the columns of adenomatous cells give the growth an alveolar appearance. There may be large multinuclear cells among the cells of the adenoma, probably due to union of pre-existing adrenal cells.

Other simple tumours, such as fibroma, angioma, lymphangioma, ganglionated neuroma, and dermoid formations have in isolated instances been met with.

Cysts are very rare in the adrenal bodies; they are small, and are probably generally due to liquefaction of adenomata. In exceptional instances cysts may result from old hæmorrhages or from hydatids. Dr. Latham has recorded a case where a dermoid cyst of the ovary infected by implantation some of the other organs in the abdominal cavity, among them one suprarenal body.

SIMPLE TUMOURS ARISING IN ACCESSORY ADRENAL BODIES OR IN ADRENAL "RESTS."—Small isolated portions of adrenal tissue, corresponding to accessory spleens, are fairly commonly present. They occur in the neighbourhood of the main glands, and appear as small yellowish-white masses. They are generally about the size of a grain of corn, and contain a medulla. They may be found along the course of the spermatic or ovarian vessels, and may travel as far as the epididymis or broad ligament. These accessory adrenal bodies may become the seat of adenomatous growth. Eurich has given a description of an adenoma starting in the medulla of an accessory adrenal body.

When accessory adrenal bodies become implanted in the substance of other organs they are called adrenal "rests." The kidneys are the most frequent situations for these "rests," but they have also been observed in the liver (Schmorl). Adenomatous growth, if it occurs in these rests, may give rise to a tumour or adenoma in the kidney, and some recorded examples of renal lipomata may be explained as the result of extensive fatty degeneration of an adrenal adenoma embedded in that organ.

PRIMARY MALIGNANT DISEASE OF THE ADRENAL GLANDS.—Primary malignant disease is rare. Some of the cases so described were very probably examples of hæmorrhagic adenomata. In a recent investigation into all the available recorded cases, Dr. Marks and I could only find sixteen undoubted instances. The criteria of malignancy that we employed were the invasion of adjacent parts, or the presence of secondary growths. To these we were able to add ten previously unpublished cases, making twenty-six in all. Taking these figures as a basis, it is seen that the sexes are equally affected by the disease; the average age was thirty-seven and a half years, being higher for males (males, forty-three and a half; females, thirty-one and a half years). The extreme ages were nine months and seventy-three years. R. Williams mentions that in thirty-six collected cases a third were in children, but I am not prepared to accept as really malignant all the cases so described. Though they have been met with occasionally in early life, there is no special tendency to these growths in infants, as there is in the case of sarcoma of the kidney.

There is often considerable difficulty in deciding dogmatically as to the exact histological nature of an adrenal tumour, which is undoubtedly malignant, as shown by the presence of secondary growths.

This is perhaps natural, since the adrenal glands may be regarded as mesoblastic from a developmental point of view, and epithelial from a morphological standpoint. Sometimes, in fact, different parts of the same tumour may resemble sarcoma and carcinoma respectively. It is interesting that the same anomaly may occur in the kidney, which has the same embryological relation to the mesoblast and the hypoblast.

Sarcoma is the form more often described, and was present in fifteen out of twenty-four cases; it may be small or large, round-celled, spindle-celled, mixed or irregular-celled sarcoma, or may contain giant cells. Carcinoma is rarer. It was present in nine out of twenty-four cases. It varies much in histological structure; the growth may resemble an adenoma, and yet, as shown by secondary growths, is undoubtedly malignant; this form may be called "malignant adenoma"; in other cases the structure is that of a spheroidal-celled carcinoma. The cells may show karyokinetic figures well, but have a special tendency to undergo fatty changes, and may contain glycogen. As the result of degenerative changes, hyaline material may be formed.

Whether histologically sarcomatous or carcinomatous, their gross anatomical characters are much the same; thus they grow rapidly, are hæmorrhagic, and have a special tendency to undergo fatty degeneration, and thus have a peculiar yellowish appearance; they break down in the centre and form cystic spaces. They may spread by direct continuity into the kidney; this is a fact I have observed myself. Targett, however, is of opinion that though flattened and mechanically displaced, the kidney is not usually invaded by the growth. On the right side I have seen the growth eat its way into the liver. Usually the growth projects forwards, but from its softness is less easily felt through the abdominal walls than a renal growth. Exceptionally, both adrenal bodies may be equally involved.

Secondary growths occur most frequently in the liver, then in the aortic lymphatic glands, lungs, and kidneys.

Clinically, malignant disease of the adrenal bodies is most likely to be regarded as a renal tumour, which is, of course, much commoner, and on review of the symptoms and physical signs it does not appear that there is any reliable method of certainly distinguishing between them. In some exceptional cases pigmentation of the skin and excessive growth of hair have been noticed, especially, but not exclusively, in young subjects with primary malignant disease of the adrenal glands; vomiting and asthenia, too, are occasionally present, but the complete clinical picture of typical Addison's disease is not presented. Emaciation and an irregular temperature are sometimes present.

Besides renal neoplasms there are other abdominal tumours, such as pancreatic cysts, hæmorrhagic abdominal cysts, retro-peritoneal lipomata and sarcomata, hydatid cysts, etc., all of which may be confused with malignant adrenal growths.

The prognosis is, like that of a malignant disease elsewhere, hopeless, unless removal is performed, and here especially it should be early and complete; the tumours grow rapidly, and generalisation may have begun before the primary growth is sufficiently advanced to have necessitated removal; this actually occurred in a case recorded by H. Morris. Osler quotes a case of completely successful removal.

Secondary growths are not uncommon in the adrenal bodies; the primary growth is sometimes in the other adrenal body. In 124 cases of carcinoma of other parts of the body examined after death in St. George's Hospital, metastatic growths were present in the adrenal bodies in fifteen, in some cases on both sides; and in forty-two cases of sarcoma of other parts of the body secondary growths occurred in the adrenal bodies in seven.

The growths always begin in the medulla of the gland, and may spread inside the capsular vein so as to project into the lumen of the inferior vena cava on the right side or the left renal vein. Both adrenal bodies may be very extensively, if not completely, replaced by malignant growth, without any clinical signs suggesting Addison's disease; in such cases the patient dies before they have time to develop.

MALIGNANT TUMOURS ARISING IN ACCESSORY ADRENAL BODIES AND IN ADRENAL "RESTS."—Structurally these tumours exactly resemble primary malignant disease of the main adrenal bodies. When the growth arises in accessory adrenal bodies situated in their ordinary position, it will give rise to a retro-peritoneal tumour; what proportion of retro-peritoneal sarcomata are derived from adrenal tissue it is impossible to say, since our knowledge on this point is at present very imperfect. Adrenal "rests" are also met with along the course of the spermatic or ovarian vessels as far as the epididymis or broad ligaments and ovaries, and in the substance of the liver (Schmorl), and may be the starting-points of malignant growths in these situations. It is, however, to malignant tumours of the kidney derived from suprarenal "rests" embedded in the kidney that attention has been chiefly directed. These tumours have aroused much interest, as they illustrate so aptly Cohnheim's theory of the origin of tumours from fœtal inclusion. Lubarsch, M'Weeney, and Targett have published valuable and critical papers on the subject. Kelynack, in thirty-two collected cases, finds that males are more often affected than females (20 to 12); the average age was fifty years for males and forty-five for females, the extremes being nine and a half months and seventy-nine years. Both kidneys are affected equally. The duration of the disease varies greatly, from a few months to ten years; the growth may remain local for a considerable period and then rapidly give rise to metastasis. Secondary growths occur in two-thirds of the cases, the lungs being the most frequent situation.

Histologically these tumours resemble the carcinomata and are composed of cells like those of the adrenal body. The same difficulties are met with in classifying these growths that have been already referred to in connection with primary malignant growths of the main adrenal bodies. M'Weeney, after an able discussion of this point, though inclining to the view that they are carcinomatous, prefers to adopt the non-committal title of "kidney tumours derived from suprarenal rests."

Clinically these tumours cannot be differentiated from renal growths. The treatment is of course removal; this has been done in several instances with success.

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Aero-Therapeutics. *See* THERAPEUTICS.

African Lethargy. *See* SLEEPING SICKNESS.

Ageusia. *See* TASTE.

Agraphia. *See* APHASIA.

Ague. *See* MALARIA.

Ainhum.—*Definition.*—A chronic morbid process confined to the toes and ultimately resulting in the spontaneous amputation of the affected part by constriction.

Geographical Distribution.—Ainhum (a negro word signifying "to saw"), on its first recognition by Da Silva Lima in 1866, was supposed to be peculiar to the negroes of Brazil. It is now known to have a much wider range, both racial and geographical, being found among Polynesians, Mongolians, Semitic races, and Asiatic Aryans. Cases have been reported from India (Lower Bengal, Orissa, Madras), Ceylon (Colombo), China (Swatow), the South Sea Islands (?), British Guiana, the southern states of the American Union, Trinidad, Madagascar (Nossi-Bé), the West Coast of Africa (especially among Kroomen), the Transvaal, Algeria, Egypt, and Syria. Our knowledge of the geographical range of ainhum is still very imperfect, and is still further obscured by the chronicity and latency of the disease, which, in consequence, may develop or be seen at a place far from the endemic area in which it was originally acquired. In some districts it is fairly common; whilst elsewhere over the vast endemic area it is but sparingly scattered. Apparently it is most prevalent in the coast regions.

We know nothing positive as to the influence of climatic conditions in the production of ainhum.

It appears to be more frequent in males than in females, in adults than in children; nevertheless, it attacks women, and occasionally has been seen in the very young.

Da Silva Lima thought ainhum had a tendency to run in families, and records instances in which several members of the same family were affected; how far this was owing to a common exposure to certain local conditions, or how far it may have depended on a common family susceptibility, it is impossible to say. Guyot described a case of what he calls congenital ainhum; most probably it was an instance of some form of congenital deformity.

Symptoms.—Ainhum usually commences as a chap at the inner angle of the digito-plantar fold of the little toe. The chap gradually extends as a slowly deepening furrow, until, surrounding the base of the toe, the two extremities of the furrow meet on the dorsal or, rather, outer surface of the member. The distal part of the toe swells, becoming round or ovoid, and looking as if the part had been constricted by a string tied tightly round the proximal end. The toe assumes a lipomatous consistency, the phalanges becoming absorbed. As a rule, the bottom of the groove is not ulcerated, but is covered by a thick layer of epidermic scales which, when removed, may disclose a somewhat raw but, usually, non-bleeding surface. Injury or neglect may lead to suppuration and ulceration at the bottom of the furrow, in which case there will be inflammatory redness, swelling and much pain.

In time the pedicle becomes so slender that it allows the swollen toe to dangle about and get in the way in walking, thereby causing pain and inconvenience. The patient then generally chops it off. The resulting wound rapidly cicatrises.

The little toe is the one usually affected, but the others may be attacked, either together (rarely) or one after the other. The constriction generally passes through the first interphalangeal articulation, but it may affect the metatarso-phalangeal joint, or even the continuity of a phalanx. It never attacks the ungual phalanx or distal articulation. It may recur in the stump of a previously partially-amputated toe. One foot may be attacked after the other.

There is a model by Baretta in the Army Medical Museum, Washington, labelled Ainhum, representing a case in which, after all the toes of the right foot had undergone spontaneous amputation, a similar process was proceeding at the middle third of the leg.

Ainhum may run its course in a few months; usually it goes on for from one to over ten years.

There are no constitutional symptoms.

Morbid Anatomy.—The horny layer of the epidermis of the entire toe is greatly hypertrophied; about the tip it may measure 1 mm. in thickness, at the seat of constriction 5 mm. In the depths of the furrow the deeper epithelial cells are swollen, and generally hyaline in appearance; occasionally they are cloudy or granular. The papillæ are greatly elongated, their capillaries being dilated and congested. The sweat-glands are atrophied, the surrounding connective tissue being increased, and the subject of an extensive fatty infiltration. There is considerable small-cell infiltration in the perivascular spaces, especially in the vicinity of the sweat-glands in the connective tissue, and in the deeper layers of the corium. There is also a notable thickening of the coats of the larger arteries, the internal coat being the subject of a proliferation similar to that found in endarteritis obliterans. The capillaries of the corium and sweat-glands are congested. The veins are usually empty: their adventitia and endothelium are swollen. There is a well-marked rarefying osteitis which advances regularly from the periosteum, becoming more irregular towards the axis of the bone. Cavities are formed in the bone which eventually become filled with a delicate reticulum of fibrous tissue and capillaries; this reticulum is filled with oil globules. Manifestly these are all secondary changes.

Etiology.—Of this nothing is known. The idea that the condition is produced artificially by the action of toe-rings, or by the tightening of a string, may be dismissed as being inconsistent with the facts. Not unlikely the process leading to the constriction begins in a crack the result of injury in walking; but most probably the established disease is a specific process induced by some germ which had obtained access to the tissues through the primary crack. Saxias of Bahia says that the wearing of boots confers no immunity. Chapping of the feet is very common among the soldiers of European nations; nevertheless ainhum is unknown among them. These are facts which suggest a specific agency confined to the endemic area of ainhum.

It has been suggested, on very meagre grounds, that ainhum is a trophic lesion connected with some nervous disorder. Collas regarded it as a manifestation of leprosy; quite recently, Zambaco has supported this view. Doubtless ainhum may concur with leprosy, and there is a resemblance in certain of the lesions of leprosy to ainhum; nevertheless the bacillus lepræ has not been demonstrated in the characteristic lesions of the latter disease.

Treatment.—When the affected part becomes troublesome it ought to be amputated. Some have suggested division of the constriction as a palliative measure in the early stages.

Air, Examination of.

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INTRODUCTION.—The condition of the atmosphere is one of the most important factors in the climate of a locality. Long before the composition of atmospheric air was known, or the researches of Lavoisier, of Priestley, and of Black had elucidated the chemistry of respiration, the importance of the atmosphere as a medium through which the health could be affected was present to the minds of physicians. Even the earliest observers attributed the origin of many diseases to variations in its condition, as regards temperature, gaseous constituents, suspended matters, etc. But only in comparatively later times, and, speaking broadly, since the beginning of this century, has its full importance come slowly to be recognised, and yet more slowly has this recognition affected practically the conditions of human life.

The percentage composition of the atmosphere, as regards its two chief constituents, varies little, and 20·9 volumes of oxygen to 79·1 volumes of nitrogen represents the result of the most careful observations. The variations in percentage composition can have little if any effect on the health; but the diminution in the absolute amount of oxygen in each inspiration in a rare atmosphere, due either to diminished pressure at high altitudes, or to increased temperature, may sometimes demand attention.

OZONE.—This allotropic modification of oxygen has been by some considered an important constituent of the atmosphere. In popular language it is oxygen in a condensed state; that is to say, while in oxygen the molecule consists of two atoms, in ozone it consists of three. Its strong oxidising power is due to the readiness with which this extra atom of oxygen breaks off in presence of an oxidisable substance. Much of its hygienic value is attributed to this action in rendering organic effluvia innocuous. Its irritating effects on the mucous membranes are due to its oxidising properties.

Schönbein, in 1840, first drew attention to this gas. In nature its most important source is atmospheric electricity. The evaporation of water is also a source; hence it is abundant in the air over the great oceans and on the sea-coast, especially during the prevalence of winds which have swept over these oceans. At high altitudes, and, according to some observers, in the neighbourhood of pine forests, it is increased in amount. It is hardly ever found in the atmosphere of cities. According to Houzeau, it is never found in greater quantity than 1 part in 700,000 parts of air. In the park at Montsouris, Paris, the mean of sixteen years' observations (1877-1892) is 0·014 milligram per 1000 litres air. The Committee of the Scottish Meteorological Society, appointed to investigate the best methods of estimating ozone, state that it was never present in the air of the Botanic Garden when the wind blew from the direction of the sewage meadows near Edinburgh. The same observers were struck with the exhilarating effects of breathing air when ozone was markedly present, and even, from their sensations, could predict when their experiments would prove its presence. Ozone was long considered as a cause of influenza, but in Dr. Parson's report to the Local Government Board on the epidemic of 1889-90 there is nothing to support this view.

The usual methods of estimating ozone are very imperfect. They are based on its power of liberating iodine from iodide of potassium, the amount of which is estimated either by the formation of the blue iodide of starch, or the depth of the brown tint of iodine, or the alkaline reaction on litmus paper of the caustic potash produced. Papers soaked in solution of iodide of potassium, with or without the addition of solution of starch, are least reliable. Papers dipped in a wine-red solution of litmus, and then for a third of their extent in a solution of iodide of potassium, as used by Houzeau, give better results. For accurate quantitative determination, probably the best method is that of aspirating the air through a solution of arsenite of potassium, and then determining the amount of ozone from the amount of arseniate of potassium produced. This is the method used in Paris.

Carbonic Acid.—Carbonic acid is a constant constituent of the atmosphere. It is present in small quantity; but, unlike the oxygen and nitrogen, its amount is liable to considerable variation. Much attention has been directed to its quantitative estimation, both by the physicist and the hygienist; indeed, it is chiefly to the simple and accurate methods devised by Pettenkofer of Munich that we must attribute the great extension of this line of investigation.

Since carbonic acid is the final product of the oxidation of the carbonaceous part of organic matter, its amount and variations in the atmosphere of a locality have been taken as a measure of the amount of such decomposition going on in that locality; that is to say, its sanitary condition. Again, since the air of occupied rooms differs from outside air in having, among other things, more carbonic acid, the amount of this gas present in such rooms is used as a measure of their ventilation.

The chief sources of the carbonic acid in the atmosphere are animal respiration, combustion of fuel, etc., decomposition of animal and vegetable matter, whether at the surface of the earth or in the soil, and volcanic and other allied phenomena. The oxidation of the organic matters (organised and unorganised) in the atmosphere itself is also not an inconsiderable source. Along with this continuous production there is a continual removal of it from the atmosphere. The means of this removal have been variously explained. Saussure attributed it to the power possessed by the chlorophyll of plants, when acted upon by light, of decomposing the carbonic acid, fixing the carbon, and restoring the oxygen to the atmosphere. This certainly is a very important means, but obviously cannot be operative during a considerable part of the year. According to Schulze, the sea plays a very important part as a regulator. When the carbonic acid attains a certain pressure (that is, a certain quantity) in the atmosphere over the sea it converts the suspended carbonates of lime and magnesia in the water into soluble bicarbonates, which are in turn reconverted into carbonates with elimination of carbonic acid, when the pressure of this gas is reduced in the atmosphere. The rôle that the soil plays, both as a source and regulator of the carbonic acid, has been pointed out by Fodor. He considers that the upper layer of the soil is the chief regulator. According as the gas is in greater or less amount in the atmosphere, the wetted surface soil either absorbs it from, or restores it to it.

The amount of carbonic acid in the atmosphere has been variously stated. The earlier researches of Dalton, Saussure, and Boussingault, which are now regarded as rather high, and the later work in this country and on the continent, give 3 to 3·5 parts per 10,000 as the average. The following are the means of daily observations at the places mentioned, in parts per 10,000:—

Paris.	Buda-Pesth.	Edinburgh.
3·26	3·88	3·52

In the streets of towns the amount is greater. The following has been found:—Edinburgh, 4·18; Dundee, 3·90; Glasgow, 4·61; Manchester, 4·42.

The seasonal variation in the amount was first pointed out by Fodor as a result of three years' daily observations. He found the maximum in autumn and early winter, and the minimum in late winter. He found another maximum in spring, and a minimum in early summer, but neither of them so well marked. The following is the result of six years' daily observations in the quadrangle of the University of Edinburgh:—

Jan.	Feb.	Mar.	Apl.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
3·33	3·64	3·48	3·41	3·34	3·35	3·39	3·44	3·45	3·83	3·88	3·38

No such variations have been observed in Paris.

Saussure found the amount of carbonic acid greater during the night than during the day. Fodor's experiments show that while this is so in autumn, in spring there is more in the morning than the evening. Similar results were got in Edinburgh. Besides these regular variations there are those of a minor degree. The amount is greater at sea than on land, and less at great elevations than at sea-level. The effect of fogs, if present, appears to be exceedingly small. If observations made on the day before a fall of rain be compared with those made on the day of the rainfall, we find, while not invariably the case, the average of the former is greater than of the latter.

The method of estimating carbonic acid in the air which was devised by Pettenkofer, is at once simple and exceedingly accurate.

It consists in absorbing the gas in a given quantity of the air by means of a solution of barium hydrate of about 7 grams to the litre. This solution must be preserved in a bottle furnished with a syphon tube by which the solution can be withdrawn, and an arrangement so that the entering air must first pass over soda lime to deprive it of any carbonic acid. By this latter arrangement the strength of the solution remains constant. The strength of this solution is determined by a solution of oxalic acid of such a strength that 1 c.c. is equal to ¼ c.c. carbonic acid at 0° C. and 760 mms. Such a solution of oxalic acid is made by dissolving 1·41 grams of the pure recrystallised acid in 1 litre of distilled water. The amount of air analysed should be not less than 5 litres, for accurate work 10 litres is necessary. For the larger quantity of air it is better to use the method of slowly aspirating it through the solution of barium hydrate contained in one or more Pettenkofer's absorption tubes placed in series. The rate of aspiration should not exceed 4 litres per hour. If the smaller amount of air be analysed, then the "bottle" method should be used. The method of collecting the sample of air varies. Into a perfectly clean and dry 5- or 6-litre bottle the air may be pumped by a bellows for about five minutes. There is here a danger of contamination with expired air of the operator. The author has found that a wide-mouthed bottle, perfectly cleaned and allowed to dry, if placed in the open air, will, in two hours, by diffusion, contain a sample of the air of the place. An extended series of observations showed that results got by this method differed quite inappreciably from the results got by the aspiration method. The mouth is now closed with an india-rubber cap, and the bottle removed to the laboratory; 100 c.c. of the solution of baryta is added, the cover replaced, and the bottle shaken from time to time during an hour, after which the solution is poured into a clean stoppered bottle and allowed to settle for a night. In testing the strength of the original baryta solution we do not examine 100 c.c. of it, but only 25 c.c., *i.e.* a *quarter* of it, and the solution of oxalic acid, as has been pointed out, is of a strength that each c.c. is equal to a *quarter* of a c.c. of carbonic acid. The number of c.c.s required is the same as would have been required if we tested the whole 100 c.c. with a solution of oxalic acid, 1 c.c. of which was equal to 1 c.c. of carbonic acid. The advantage of this is obvious when we come to examine the solution of baryta that has absorbed the carbonic acid from the air, as we can make three separate tests or titrations at least from every experiment, and thus be able to check the work.

Example:—

25 c.c. original baryta solution	=42·5 c.c. oxalic acid solution.
„ after absorption of carbonic acid	40·1
Amount of carbonic acid in air examined	2·1 c.c.
Capacity of bottle . . .	5400 c.c.
Deduct bulk of baryta solution added	100
	5300 c.c. = amount of air examined.

The volume of air is now corrected for temperature and pressure by the well-known calculation, and the amount of carbonic acid calculated in 10 litres, *i.e.* volumes per 10,000 volumes, at 0° C. and 760 ms.

Ammonia.—Ammonia or ammoniacal salts are present in very small quantity in the atmosphere. The following results are per 1000 litres:—

	Buda-Pesth.	Paris.	Edinburgh.
Mgrms. . .	0·03	0·024	0·029

Fodor found it highest in summer and lowest in winter. He considers that the amount of ammonia present in the air of a locality is a measure of the amount of decomposition of animal matter taking place at the surface. At a time when the air of the quadrangle of Edinburgh University contained as a mean 0·021 mgrms. per 1000 litres, the atmosphere of a restricted area of the city, which was being raised by the deposit of building and other rubbish, and where therefore there was a good deal of organic decomposition taking place, contained as a mean of four experiments 0·13 mgrm., or about six times the amount. The following results per 1000 litres obtained in Glasgow show the increase in the amount of ammonia in the atmosphere on passing from the outskirts to the centre of a great city:—

Western Infirmary	0·015 mgrms.
Hospital, Kennedy St.	0·019 „
Sailors' Home	0·024 „
Calton	0·044 „
Stirling Square	0·053 „

In the quantitative estimation of the ammonia in the atmosphere a large volume of air must be examined, and in no case should it be less than 250 litres. In the Edinburgh observations 1000 litres are examined in each experiment. The air is aspirated through ammonia-free water, and the amount of it determined by Nessler's reagent. A better though more laborious method is to aspirate through weak solution of hydrochloric acid the amount of ammonia in which has been previously determined. By neutralising this with ammonia-free caustic alkali and then distilling, the amount of ammonia is determined by Nessler's reagent in the distillate. On deducting the amount of ammonia in the acid used, the remainder is the amount of ammonia in the air examined.

Moisture.—Water vapour is a constant constituent of the atmosphere, but its amount is subject to great variations. When air at a given temperature contains as much water vapour as it can take up it is said to be saturated at that temperature, that is to say, if the temperature be lowered water will be deposited, while if the temperature be raised, the air ceases to be saturated and must take up more water in order to be saturated at the new temperature. The temperature of such saturated air is called its dew point, and for each such temperature there is a corresponding pressure of water vapour. In all books on meteorology there is given for each temperature the corresponding amount of water vapour in an atmosphere saturated at that temperature, expressed either in terms of the pressure it is exerting or of its weight in grains per cubic foot or grammes per cubic metre. The actual amount of water vapour in a given atmosphere is termed its *absolute* humidity, and is expressed in either of the ways abovementioned. The relative humidity is the relation between the amount of water vapour actually in the atmosphere and the amount of water vapour it could contain if it were saturated at the temperature, and is always expressed in percentages, complete saturation being 100. For example, an atmosphere whose relative humidity is 75 contains three-fourths of the amount of water vapour it could contain if it were saturated. Relative humidity is said by some to express the drying power of the air, that is, the amount of water vapour it can still take up. This is only true if, together with the relative humidity, the temperature of the air is also given, and even then it is not the clearest or simplest way of expressing it. If we compare two atmospheres, both with 75 per cent of relative humidity, but the temperature of the one is 50° F. and of the other 70° F., the drying power of the former is only one-half that of the latter. This drying power, as Flügge first pointed out, is better expressed by the term "defect of saturation," that is, the difference between the pressure of water vapour at the temperature of the dew point and at the temperature of the air. Still the atmosphere may have a great drying power though the defect of saturation is small. Middendorff states that in East Siberia, with the temperature much below freezing point, where therefore both the absolute humidity and defect of saturation were insignificant, the drying power was such that fur coats made moist by perspiration when spread on the snow at night were quite dry in the morning.

There is more water vapour in the atmosphere at the tropics than at the poles, more at sea than on land, and the amount on land diminishes on going inland. It is greater in summer than in winter, and at mid-day than at morning and night. Proximity to large sheets of evaporating surface, whether marshes, lakes, or seas, associated with high temperature and absence of wind, are the conditions which produce a high amount of atmospheric moisture. But these are much affected by winds. At Cape Juby, on the extreme east coast of Morocco, where the conditions of high atmospheric moisture are present, when the strong hot east wind from the desert is blowing, the relative humidity may fall to 18 per cent. Madeira and the Canary Islands are affected by a similar wind called Leste or east wind. In Funchal, 80 miles from the African Coast, the relative humidity has been observed to fall to 20 per cent. The absolute humidity of the atmosphere rapidly decreases on ascending high mountains. This decrease is more rapid than the decrease of atmospheric pressure. The relative humidity is little changed. At great elevations in the Alpine districts the atmosphere is driest and the sky freest from clouds in winter. The drying power of the air is very great at this season and is dependent not only on the low relative humidity but perhaps chiefly on the diminished pressure favouring evaporation. Dead animals mummify rather than putrefy, and in the lower Engadin air-dried flesh is a common article of food among the people.

Both indirectly and directly the atmospheric moisture has an important influence on health. When in the form of droplets, as clouds, it certainly reduces the effects of solar and of terrestrial radiation, and thus lessens the daily range of temperature in hot regions. In high altitudes, such as Davos, during the cloudless weather of winter, the temperature in the sun as registered by the black bulb thermometer gives a mean of 86° F. in December, with a maximum air temperature below freezing point. It is to this circumstance, together with the dryness of the atmosphere and the absence of wind, that Davos owes its fame as a winter health resort. Whether the water vapour in the atmosphere has this effect of absorbing heat rays is a point on which authorities differ, but all are agreed that in a dry atmosphere a much higher mean temperature can be borne than in a moist one. The evaporation of moisture from the skin is the great regulator of the temperature of the body, and if the moisture in the air be great, this evaporation is interfered with. The great amount of moisture in the atmosphere of tropical and subtropical places situated on the coast is an important cause of the harmful effects of the high temperature.

There are several methods of determining the amount of water vapour in the atmosphere, but only two can be described here—

1. The Hair Hygrometer.—This gives the relative humidity. For rapid observations this instrument is useful, though its readings are not always trustworthy. Its action depends on the extension produced on hair when it is moistened. A long hair which has been carefully freed from fat, is stretched and connected with a pointer which travels on a scale and indicates the lengthening or shortening of the hair. The degrees on the scale are not proportional to the relative humidity, so that each instrument must be graduated against a more exact one, such as is next described.

2. The Wet and Dry Bulb Thermometer.—From the readings of this instrument the dew point can be calculated. It consists of two thermometers. The bulb of one is surrounded by muslin, from which passes a few strands of darning cotton into a vessel of distilled water. The muslin and cotton must previously be cleansed from any fatty matter. After wetting the muslin on the wet bulb it is kept continually moist by the water rising by capillarity through the cotton. In performing an experiment the apparatus is allowed to stand in the air for about one hour. (When observations are regularly made, this will form one of the instruments in a Stevenson's louvred screen, and therefore will always be exposed.) If the air is saturated with moisture then both wet and dry bulbs will show the same temperature. If not, the wet bulb will be lower than the dry bulb, and the drier the atmosphere the amount of difference between them will be greater. Evaporation takes place from the muslin, and the heat required for this is drawn from the bulb. Dr. Apjohn gave an elaborate formula for finding the dew point from these two readings. Mr. Glaisher has given a simple method, which obviates the need of this calculation. By an extended comparison of the readings of the wet and dry bulb thermometer with the dew point as otherwise determined, he has

provided a series of factors by the use of which the dew point can be readily got from these readings. The rule is—multiply the difference between the temperature of the wet and dry bulb thermometers by the factor opposite the temperature of the dry bulb, and subtract the product from the temperature of the dry bulb thermometer. This gives the dew point. From the dew point we find the absolute humidity (f) by consulting tables. If the pressure of water vapour at temperature of dry bulb, *i.e.* of the air, be s, then

$$\frac{f}{s} \times 100 = \text{Relative humidity}$$

and $s - f$ = defect of saturation.

Micro-organisms.—The micro-organisms found in the atmosphere are almost invariably non-pathogenic. Both moulds and bacteria are found, the latter as a rule predominating. Of the moulds Penicillium glaucum is most frequent, but aspergillus and mucor are by no means rare. Of bacteria the micrococci are most frequent, the chromogenic forms, *e.g.* cinnabareus, aurantiacus, Sarcina lutea and S. rosea predominating. Of bacilli, subtilis and mesentericus are not uncommon. Chromogenic saccharomyces are also found. The quantitative estimation of the micro-organisms in air has been made by various observers, but by none so systematically as by Miguel in Paris. His observations are made both at Montsouris in the outskirts of Paris and in the centre of the city. The following shows the number per 1000 litres arranged as to season and place:—

	Montsouris.		Centre of Paris.	
	Moulds.	Bacteria.	Moulds.	Bacteria.
Winter	145	170	1345	4305
Spring	195	295	2275	8080
Summer	245	345	2500	9843
Autumn	230	195	2185	5665

Any relation between the number of micro-organisms in the atmosphere and the incidence of epidemic disease has not been made out. The nearest approach to such a relation is recorded by Miguel. In 1893 he found that on four separate occasions a sudden increase in the number of micro-organisms in the air was followed some weeks later by a sudden increase in zymotic mortality. The importance attributed by some to the air as a medium of spread of pathogenic organisms has not been verified by bacteriological investigation. The bacteria found in the atmosphere are carried on particles of dust. The pathogenic organisms which cannot live in the dry state (non-spore bearing) can only live on particles of dust sufficiently large to prevent complete drying, and these particles, owing to their size, are only transportable by the air for very short distances. According to Germano, infection of typhoid fever by dust is impossible at a distance of a few hundred yards. But in the case of diphtheria this is possible at greater distances, as the bacillus of Löffler preserves its full virulence when quite dry. The effect of light, also, in either killing the organisms or robbing them of their virulence, renders infection at great distances unlikely.

Sewer Air.—The air of sewers has long been regarded as dangerous and a likely source of infection in certain diseases, but it is only of recent years that much attention has been directed to its careful examination. The gaseous products of the decomposition of animal organic matter have been sought for, as also the micro-organisms of infectious disease, *e.g.* the bacillus of typhoid fever. The investigation carried out in 1893 by Mr. J. Parry Laws for the County Council of London is, perhaps, the most extensive of its kind in this country. As a mean of thirteen experiments the average amount of carbonic acid was 11·8 parts per 10,000; and the number of micro-organisms was, with two exceptions, less than in outside air. The kind of micro-organisms found was the same as those in outside air. The Bacillus coli communis and the Micrococcus ureæ, both abundant in sewage, were carefully searched for, but never found. In a later investigation of the character of the microbes in sewage, carried out along with Dr. Andrews, they found that the very organisms most abundant in sewage are precisely those absent from sewer air. Their conclusion is—"That the air of sewers themselves plays any part in the conveyance of typhoid fever appears, as the result of our investigations, in the highest degree unlikely." Carnelly and Haldane conclude, from their investigation of sewer air in London and in Dundee, "that the air of sewers, as regards organic matter, carbonic acid and micro-organisms, is in a very much better condition than the air of naturally ventilated schoolrooms." The only pathogenic organism found by Uefelman in sewer air was Staphylococcus pyogenes aureus.

In Paris the air of sewers is regularly examined. The following are the results for 1892:—Carbonic acid, 4·78 per 10,000; ammonia, ·148 mgrms. per 1000 litres; the outside air containing 2·98 parts per 10,000 carbonic acid, and ·04 mgrms. of ammonia per 1000 litres. A litre of sewer air and of outside air contained 6 and 8 micro-organisms respectively.

GROUND AIR.—The chemical examination of ground air (Grund Luft of Pettenkofer), *i.e.* the air which is contained in the pores of the soil, was first made by Boussingault and Levy in 1853. Not much attention, however, was directed to it till Pettenkofer showed that the amount of carbonic acid in the air of a given soil could be used as a means of estimating the amount of organic decomposition taking place in it. Ground air is simply atmospheric air with its oxygen in part replaced by carbonic acid, with the occasional presence in small quantity of some other gases—carburetted hydrogen and ammonia. Boussingault states that when the sum of the amounts of oxygen and carbonic acid exceeds the amount of oxygen in the atmosphere, then *putrefaction* is taking place in the soil. In this case, under the action of anaerobic organisms, the organic matter is itself supplying the oxygen for the oxidation of its carbon. In the deeper layers of an impure and rather impermeable soil, putrefaction is common if the suitable conditions of warmth and moisture are present; in an impure and permeable soil, on the other hand, there is simply oxidation.

The amount of carbonic acid in the air of a given soil depends on (1) the amount of organic matter; (2) permeability; (3) depth; (4) temperature and moisture. Pettenkofer found that the ground air in the desert of Sahara had the same composition as the atmosphere, the conditions for the development of carbonic acid being absent. But no reliable conclusions can be drawn as to the purity of a soil from the carbonic acid estimation, unless its permeability is known. If the ground air in two soils be examined, similar as regards organic impurity, temperature, and moisture, but of different permeabilities, the less permeable will contain more carbonic acid than the more permeable; because, in the former, the air is prevented from so easily diffusing vertically into the atmosphere, or horizontally into the adjacent soil. As regards temperature, the amount of carbonic acid is least in late winter and spring, increasing to its maximum in summer and early autumn, and declining again to its minimum. The rainfall exerts a great influence, especially if rain follows on a long drought in summer. The amount of carbonic acid increases with the depth. This arises, not from increased chemical change taking place, nor necessarily from more organic impurity being present, but because the air does not get so easy vent as in the more superficial layers. The following are the monthly means of daily determinations at a depth of 3 feet. (1) is an impure and permeable soil, and (2) a pure and rather impermeable clayey soil. The results are stated in c.c.'s per litre.

	Jan.	Feb.	March.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
(1)	8·68	7·2	7·66	7·9	9·0	15·6	24·9	24·6	20·4	17·6	11·6	8·4
(2)	4·1	4·5	3·9	4·5	4·1	4·9	6·4	8·3	8·0	8·5	3·9	3·2

Lewis and Cunningham investigated the ground air in the soil of Calcutta. They found the amount of carbonic acid at 3 feet as follows:—

Jan.	Feb.	March.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
6·2	5·6	5·6	4·7	4·1	5·6	4·0	10·2	11·3	9·7	8·8	7·0

Lewis and Cunningham consider the cause of the seasonal change in the amounts of carbonic acid to be the ventilation of the soil, and that this change is an index of the degree in which emanations escape from the soil into the atmosphere at different times of the year. The sudden increase in August is coincident with the high rainfall. According to these observers, the rain acting on a finely-textured, loamy soil, such as is found in Calcutta, closes the pores of the soil and prevents the ventilation going on. Hence the fall in spring is due, not to diminished production, but to increased escape of the carbonic acid into the atmosphere. The relation of the changes in composition of the ground air and incidence of cholera is striking. November, April, and August are the months of medium, maximum, and minimum cholera prevalence. Both in Buda-Pesth and in Edinburgh there is a marked relation between the rise of the amount of carbonic acid in the ground air and epidemic diarrhœa of children.

LITERATURE.—1. FODOR. *Luft Boden und Wasser.*—2. HANN. *Klimatologie.*—3. CORNELIUS FOX. *Ozone.*—4. *Annuaire de l'observatoire de Montsouris.*

Albinism. *See* SKIN.

Albuminuria and Albumosuria. *See* URINE.

Alcohol (SYN.—Spiritus Vini).—*Ethylic Alcohol* is obtained from absolute alcohol by the action of sodium or caustic lime. It has also been derived synthetically from coal, and even built up from its elements. In the pure anhydrous form it is a hygroscopic inflammable fluid of sp. grav. 0·80625, of vap. D 1·6133, boiling at 78°·3 C., viscous at – 100°, and freezing at – 130°. For commercial purposes it is derived from starches and sugars by fermentation, distillation, and rectification. The by-products of this process are difficult to remove, fusel oil always occurring in the distillate from starches as a mixture of amylic and other alcohols. Fatty acids and ethereal salts may also remain, and a small amount of furfurol, a convulsant poison, may be present. A knowledge of these by-products is important, as their presence is frequently the cause of the toxic symptoms seen in alcoholism.

Tests.—The presence of alcohol is detected by the "iodoform test" (a few crystals of iodine warmed in the sample with just enough KHO to decolourise the iodine); this, however, also reacts with bodies of the general formula $CH_3.CO.C$, or $CH_3.CH(OH).C$. The "ethyl benzoate" test (benzyl chloride and warmth), the "acetic ether" test (sodium acetate, sulphuric acid and warmth), and the "chromic acid" test (a small crystal and warmth give a green colour) are not conclusive singly. The guaiacum and the molybdic acid tests described by Elias H. Bartley are delicate tests. To a filtered extract of guaiacum powder a few drops dilute HCN solution and a drop of weak $CuSO_4$ solution are added. If 1 per cent or more alcohol be present, a blue colour is produced.

A solution of 1 part of molybdic acid in 10 of strong H_2SO_4 is allowed to run down the side of the test-tube containing the suspected liquid. If alcohol, ether, or aldehyde be present, a blue ring will form at the junction of the two liquids. This will detect 0·1 per cent alcohol.

Spirits.—The alcoholic strength of spirits is as follows:—

Absolute Alcohol	95·5-99·5 per cent.
Rectified Spirit	90 per cent.
Proof Spirit	57·09 per cent by volume, or 49·3 per cent by weight.[1]
Methylated Spirit	Pure form—90 per cent alcohol of density 0·830 and 10 per cent purified wood spirit. Common form—contains in addition petroleum oil.
Genuine Brandy or Cognac	Distilled from wine, contains œnanthic and other ethers—35-45 per cent.
Rum	Distilled from molasses, contains butylic ether—50-70 per cent.
Gin or Hollands	Flavoured with juniper and sugar —50-60 per cent.
Whisky	Needs mellowing to remove excess of fusel oil and traces of furfurol—50-60 per cent.

German spirit is obtained from potato, Japanese spirit from rice or sorghum, Arak from cocoa-nut or palm juice.

Sweet liquors and bitters are compounded from alcohol and aromatic essences.

Mead obtained from honey, and koumiss from mare's milk, are solitary instances of the alcoholic fermentation of animal products.

Genuine wines are the product of a spontaneous fermentation of the juice of the grape, and in the case of sparkling wines of a secondary fermentation of added sugar. Some are sold which are wholly artificial, others are only partly derived from the grape. They are liable to various manipulations, "blendings," and "flavourings" with ethers, etc., to imitate definite brands and vintages. Some are fortified, and many French, Spanish, and Italian wines are "plastered," tartrate of lime being precipitated, and potassium sulphate, which is said to produce cirrhosis of the liver, remaining in solution, whilst the calcium carbonate accompanying the gypsum precipitates any free tartaric acid, leaving malic acid only as the representative of the original acidity.

WINES.—Wines may be classified as—(1) spirituous wines, (2) sweet or liqueur wines, (3) clarets or Bordeaux, (4) Burgundies, and (5) Rhine or Moselle wines.

Their Constituents.—All wines contain colouring matters, and subtle principles which give flavour, bouquet, and aroma. Their further composition may be tabulated as follows:—

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[1] For the purposes of excise, the strength of alcohol in solutions is compared with proof spirit, not alcohol, and estimated in percentages or degrees above or below proof. Proof spirit should weigh exactly $\frac{12}{13}$ths of an equal measure of distilled water.

Alcohol	7 to 20 per cent.
Acids	Tartaric, Malic, Succinic, Butyric, Hydrochloric, and Sulphuric.
Sugar, traces only; or	1·5 to 9 per cent in sweet wines.
Extractives	1·5 to 10 per cent.
Tannin	From the skins and pips or from the cask.
Glycerine	0·2 to 0·6 per cent.
Mucin and Albumen	Traces only.
Ash	0·2 to 0·4 per cent.

The acids are partly combined with potassium, sodium, calcium, magnesium, aluminium, and iron.

THE AVERAGE PERCENTAGES OF ALCOHOL, OF SUGAR, AND OF ACIDITY IN WINES ACCORDING TO KÖNIG (QUOTED BY VON LEYDEN).

	Alcohol by Weight.	Acidity reckoned as Tartaric Acid.	Sugar.	Extractives.	Ash.
Moselle	12·06	0·61	0·20	1·88	0·20
Rhine Wine	11·45	0·45	0·38	2·23	0·17
Hungarian White Wine	8·00	0·69	0·07	2·33	0·20
French Red Wine	9·40	0·59	0·62	2·34	0·22
Port Wine	16·41	0·47	3·99	6·17	0·29
Madeira	15·60	0·49	3·28	5·28	0·31
Sherry	17·01	0·53	1·53	3·47	0·46
Champagne	9·22	0·58	10·70	11·20	0·14

The *free acidity*, reckoned as tartaric acid, varies between 0·2 per cent to 0·4 per cent in the better wines and 0·7 per cent in the inferior. Wine containing less than 0·6 per cent is dull; but with more than 1 per cent it becomes unpalatable (von Leyden).

Beer differs from wine and other alcoholic liquors in its high nutritive value. In a litre of beer there is as much albumen as in 120 grammes of milk, and as much carbohydrate as in 100 to 150 grammes of bread (von Leyden). Again, fermentation, which is due to the yeast-cells, or more probably, as recently demonstrated by Wróblewski, to their ferment or enzyme, is checked before the carbohydrate is completely split up into its terminal products alcohol and carbonic acid.

The constituents of beer are:—

Water	90 per cent.
Alcohol	3 to 5 ,,
Organic acids—lactic, succinic, propionic, and traces of acetic—reckoned as lactic acid: 0·116 per cent in German to 0·31 per cent in English beer.	
Sugar	0·40 to 0·90 per cent.
Maltose and dextrose	4 to 5 ,,
Albumen	0·37 to 0·8 ,,
Extractives	4·99 to 7·23 ,,
Carbonic acid	0·1 to 0·2 ,,
Ash	0·18 to 0·28 ,,
Small quantities of glycerine, and of the aromatic, oily, and bitter principles of hops, including lupulin.	

It will be seen that beer is less acid than wine, although its large percentage of carbohydrate may give rise during digestion to greater acidity.

German beers are lighter as well as less alcoholic than those brewed in this country. Their percentage of extractives by weight is estimated at 4·82 for Pilsener beer, and at 5·8 for ordinary Munich beer, against 6·8 for London porter, and larger amounts for Burton ale and for Scotch ale. This explains their greater digestibility and their smaller tendency to produce gout and an abnormal deposition of fat. Their fermentation conducted at a low temperature and other differences in their mode of production may also partly account for the fact that they are better tolerated by delicate stomachs.

Cider varies greatly in its proportion of alcohol and sugar, according to the degree of fermentation which it has undergone. Rough or hard cider is the more highly fermented variety. *Perry* is analogous to cider. Von Leyden gives the following percentages for cider:—Alcohol, 4·72; acidity (reckoned as malic acid), 0·54; sugar, 0·21; extractives, 2·34.

TABLE OF THE ALCOHOLIC STRENGTHS OF VARIOUS WINES AND BEERS (ROSCOE).

Port (old bottled)	20·2 per cent.
Port (newly bottled)	17·4 „
Montilla sherry (1854)	16·3 „
Fine Marsala	17·0 „
Madeira	16·1 „
Beaune	13·5 „
Oberingelheimer	9·4 „
Rudesheimer	9·2 „
Assmannshauser	9·1 „
Château-Lafite	8·7 „
Ordinary Bordeaux	6·4 to 8·7 „
Johannisberger (1842)	8·1 „
Auerbacher	8·4 „
Burton ale	5·9 „
Edinburgh ale	5·7 to 6·1 „
London porter	5·4 to 6·9 „
Munich lagerbier	5·1 „
Schenkbier	3·8 „
Berlin weissbier	1·8 to 2·0 „

THE PHYSIOLOGY OF ALCOHOL

GENERAL PHYSIOLOGY.—*Action upon cells, metabolism, ferments, and bacteria.*—*Paralysis* is the primary effect of alcohol upon protoplasm, which it deprives for a time of its irritability, contractility, and sensibility, probably by dehydration. *Metabolism* is checked as a secondary result. All cells are affected, and none so much as the oxygen-carriers; but, unlike hydrocyanic acid, a yet stronger cell-paralyser, alcohol only partially arrests the power of the red blood corpuscles to attract and to yield up oxygen. Nerve-cells are specially susceptible in varying degrees; and the function of glandular cells is readily inhibited.

Ferments are not destroyed, but carried down from their solutions by absolute alcohol with the coagulated albumens, or locked up in the hardened and shrunken tissues immersed in it, and may be kept indefinitely for future use. Artificial digestions are arrested.

Bacteria exhibit in different measures a remarkable power of recovery from their temporary alcoholic paralysis, in the case of some of them, even when this is due to immersion in boiling alcohol. A microbe has been discovered which grows in "faulty" rum. The addition of alcohol to impure water probably does not correct its infectiveness, though it may favourably modify the liability of the individual to infection.

THE FATE OF ALCOHOL IN THE ECONOMY.—(1) *Its absorption.*—Alcohol is absorbed by mucous membranes, rapidly by that of the stomach and intestine, readily also by the respiratory mucous membrane when it is injected or inhaled into the trachea, by the blood-vessels and lymphatics when it is injected subcutaneously, and by open wounds. Its absorption through the unbroken skin is regarded as doubtful (Richet).

(2) *Its combustion.*—According to Jaillet, acetic acid is formed in the red discs and rapidly burnt to CO_2, and aldehyde, one of the by-products, may be recognised in the breath by its smell, whilst in the stomach dilute alcohol is partially converted into acetic acid and acetates—a change which is impeded by concentration or by alkaline fluids. Hence water is a better check upon inebriation than alkaline table waters (Richet).

In health, small supplies of alcohol may be completely oxidised into CO_2 and H_2O with the exception of fractional quantities excreted by the kidneys and by the lungs. So long as this result obtains, the so-called "physiological amount" (estimated at about 1½ oz. of alcohol, and equal to three ounces of whisky or one pint of claret, or one and a half pints of ale for a robust man) has not been exceeded. Under abnormal conditions larger amounts may be burnt off in the economy.

(3) *Its elimination.*—Beyond this physiological allowance a larger proportion, 3 to 5 per cent, but according to Strassmann as much as 10 per cent, is excreted unchanged. Binz allots an excretion of 2·91 per cent to the kidney, of 1·60 per cent to the lung, and of 0·14 per cent to the skin. Alcohol does not seem to be eliminated by the intestine.

SPECIAL PHYSIOLOGY. — *The actions of alcohol upon the organs and functions.*—The action of alcohol varies much with its dose and concentration. Between its "minor" effects, sometimes called "physiological," and its "toxic" effects there is no strict demarcation. The action varies also with individual susceptibility, which may amount to an idiosyncrasy.

Digestion.—Congestion, which may in the case of concentrated alcohol lead to ecchymosis, is accompanied with increased peristalsis and a feeling of warmth. The glandular secretions which small amounts stimulate are arrested by large and strong doses; but even a moderate proportion of alcohol perceptibly retards their digestive action. Thus peptic digestion is slowed by 2 per cent, greatly slowed by 10 per cent, and almost suppressed by 15 per cent solutions (Schulz), and the activity of ptyalin is similarly reduced (Aitchison Robertson), though less markedly than by tea. The duodenal, pancreatic, and intestinal digestions suffer secondarily. The *appetite* is diminished or suppressed and assimilation is checked.

Circulation.—Quickening and increased force of cardiac action are the initial but temporary results of the stimulation of the heart and of its excito-motor ganglia by alcohol. This is accompanied by a relaxation of the blood-vessels, which is conspicuous as a cutaneous flush, but is not limited to the skin, and is due either to active dilatation (Binz) or to vaso-paralysis (Schmiedeberg). The systole of the heart is hurried as well as forcible, and its diastole is curtailed. These peculiarities are reflected in the characteristic alcoholic pulse, with its tall and abrupt but short wave, and with its sudden loss of tension. The result is a greater speed of the circulation and a lessened venosity of the blood; and the nervous system is "stimulated" by an increased supply not only of arterial blood, but of the alcohol which it conveys. Flagging energy is revived, depression is changed to brightness, cerebration is quickened, and warmth restored to chilled surfaces. But at a later stage the conditions are reversed, the blood circulates more slowly, its respiratory interchanges are diminished, and it contains relatively more carbonic acid.

Respiration.—The respiratory rate is accelerated at first, and there may be some increase in the depth of breathing secondary to the general excitation; but hyperpnœa is not a feature of alcoholic poisoning. Later, respiration becomes slower, and, under the influence of excessive doses, laboured, stertorous, and irregular till it finally ceases. Early arrest of respiration, such as that caused by prussic acid, is exceptional.

Metabolism and its Products: Heat and Energy.—Alcohol obviously acts as a fuel in the proportion in which it is oxidised, and to that extent it may save from oxidation some of the fat of the tissues and possibly some of their albumen (Fokker and J. Munk). The marked decrease in the urinary phosphates and sulphates and in urea, whilst uric acid is largely increased (Chittenden), is, however, generally ascribed less to this vicarious combustion than to the depression of metabolism by alcohol. According to J. Munk, in the special case of large doses administered to animals this depression is preceded by an initial increase in the nitrogenous catabolism coincident with the stage of excitement and of agitation, which also occasions an increased expenditure of oxygen (von Boeck and Bauer). But with the onset of confirmed intoxication the oxygen intake drops by 13 per cent, the production of CO_2 by 12·4 per cent, and the heat production by 19 per cent (Deplats, quoted by von Leyden).

Temperature, Thermogenesis, and Thermotaxis.—Alcohol restores body heat in chill and in collapse, but fails to maintain it, as it wastes by increased radiation part of the warmth which it supplies. A worse fault is that it soon checks its own combustion as well as that of other heat-producers. Soulier ascribes to it a direct temperature-lowering action upon the central nervous system, although a considerable rise in temperature has sometimes been recorded in acute poisoning by alcohol. Perhaps also the fine mechanism of heat-regulation may suffer in its peripheral conduction. A blunted appreciation for external temperature often leads in intoxication to a continued exposure to cold; but the depression of the temperature is usually the combined result of a lessened production and of an increased expenditure of heat. Chilliness succeeds to the preceding glow, and in severe intoxication algidity supervenes. Hence the special danger of alcohol in intensely cold atmospheres.

The production of energy or the capacity for work, muscular and nervous, rises and sinks with the cardio-vascular efficiency. Whilst the heart is cheered more work may be done with less effort, but as cardiac depression supervenes, the power for work falls to a lower level than before. Apart from the influence of their blood-supply the nervous and the muscular cells are obviously impaired by alcohol as generators of energy—and for feats of endurance alcohol has to be laid aside. The influence of alcohol upon the *higher nervous centres* will be considered under the heading of "Alcoholism."

The Renal and the Cutaneous Functions.—Much of the diuretic action sometimes attributed to alcohol is due to the water with which it is diluted, or to added diuretic principles. Before it reaches the kidney the blood has been freed of a large proportion of the alcohol it had absorbed; and, moreover, any direct stimulation of the secreting epithelium would be counteracted by a lessened general arterial pressure. Diaphoresis is not a prominent result, in spite of the cutaneous hyperæmia, except when alcohol is taken with hot water

The generative function is depressed after a temporary stimulation. Inebriation is credited with a marked influence upon the products of conception, individuals conceived during acute alcoholism being often degenerate, and Féré has succeeded in producing monsters by the influence of alcoholic vapour on eggs (Dubois).

TOXIC EFFECTS OF ALCOHOL AND THEIR CLINICAL MANIFESTATIONS

THE VARIETIES OF ALCOHOLISM.—I. The *acute toxic* conditions immediately due to alcohol are of two kinds—(1) common alcoholic intoxication, and (2) acute alcoholic poisoning. The symptoms in both cases are almost exclusively nervous. II. *In chronic alcoholism*, by the side of the functional symptoms which are the remote effect of alcohol, there is always some structural change or degeneracy. III. *In delirium tremens* the nervous symptoms are acute and yet not due to alcohol itself, of which not a drop may be in circulation. It is essentially an acute failure of nerve power supervening upon prolonged alcoholic malnutrition.

Individual susceptibility varies, and idiosyncrasies occur which are often more marked for special kinds of intoxicating beverage than for alcohol itself. *Resistance* is increased by habit, but at last it is lowered; and the beginning of delirium tremens is often an inability to imbibe the accustomed stimulating dose. This intolerance of the debilitated drunkard is often inherited by his progeny, in whom it may be associated with other idiosyncrasies. An opinion prevails that the tendency of this age is towards a greater susceptibility to the toxic effects of alcohol.

An *immunity* from the usual alcoholic intoxication is witnessed in profound disturbances of the nervous system, such as intense pain, shock, strong emotions, and high fever; its mechanism is ill understood.

Pure ethylic alcohol intoxication is seldom witnessed; it is usually modified by the ethers of wine, by the higher alcohols of spirits, and by the special ingredients in other alcoholic drinks. The toxic properties of the higher alcohols are said by Rabuteau to increase with their molecular weight and boiling-point, and Richet draws attention to the influence of their solubility and volatility upon the kind and duration of the resulting intoxications. Natural wine being free from them, though it will produce inebriety, does not, unless taken in very large quantities, lead to chronic alcoholism: it is mainly against the consumption of spirits that legislation and fiscal checks need to be directed (Richet).

The convulsive element which sometimes complicates intoxication by spirits is the most obvious instance of a mixed intoxication. The oil of wormwood contained in absinthe (0·33 per cent) acts as a convulsive poison upon the cortex, the motor centres, and the medulla; and the chronic degeneracies due to it are also obviously distinct from simple alcoholic poisoning (*vide* "Absinthism"). Ether intoxication also differs from that produced by alcohol. The basis of most compounds used surreptitiously as intoxicants, such as Eau de Cologne, various medicinal tinctures and tooth washes, is essentially alcoholic.

The deleterious effects, both acute and chronic, of the malt liquors are also instances of a mixed intoxication varying with their constituents; and the pathological results differ from those due to spirit-drinking. In different degrees their habitual use promotes a deposition of fat.

ACUTE ALCOHOLIC INTOXICATION AND ITS STAGES.—This is described in the article "Alcoholism."

The nervous lesions which are characteristic are described in the article

"Alcoholism," and only the visceral lesions that may be present will here be referred to.

THE MORBID CHANGES IN CHRONIC ALCOHOLISM

CHRONIC ALCOHOLISM.—The pathological appearances in cases of chronic alcoholism vary with the individual, but we trace in all of them the influence of irritation and of malnutrition.

Changes of Malnutrition.—The degeneracy which overtakes all tissues, from the integuments which become peculiarly thin and soft to the skeleton with its undue lightness and fragility of bones, may be disguised at first by an increased storage of fat, but it tends towards atrophy, particularly in spirit-drinkers. Fatty decay is the leading alteration in muscle, blood-vessels and glandular cells alike, whilst fatty deposition and replacement-fibrosis go hand in hand with parenchymatous atrophy. The liability to tuberculosis at periods of life which are otherwise almost immune demonstrates the lowered resistance of the organism and of its constituent elements.

The direct irritative changes affect chiefly the upper alimentary tract, the liver, the heart, and the blood-vessels, which are immediately exposed to the poison, and the nervous tissues which are the most sensitive to it.

In alcoholic pharyngitis, œsophagitis, and gastritis congestion is associated with the changes which accompany chronic catarrh. Thickening of the gastric mucous membrane with slight fibrosis is produced by ardent spirits, whilst dilatation of the stomach may be the mechanical result of the beer-drinker's libations.

The frequency of the two forms of *cirrhosis of the liver* is less surprising than the fact that they are not more constant and earlier to appear. Some livers may be tolerant of the poison, others exceedingly vulnerable. Chronic excesses in beer and in spirits are the primary causes; but the mechanism of the change needs further elucidation.

The kidney probably suffers slight structural lesions to which the transient attacks of albuminuria may be due. An increased bulk of the organ is, however, usually more obvious, particularly in the beer-drinker's kidney, than any qualitative change. Beer-drinking may nevertheless, by promoting gout, lead to granular kidney.

The Blood-Vessels and the Heart.—Atheroma may be associated with the simple fatty changes which occur in the walls of the aorta, and of the larger arteries; and the arterioles are apt to be thickened. The veins are frequently large and relaxed; and piles are common in cirrhosis. The heart may present no change beyond increased softness and friability of the myocardium, due to fatty degeneration. In some cases the extreme changes described by Graham Steell under the name of the "alcoholic heart" are found. Though the kidneys are not granular but large and congested, the heart in this condition is considerably hypertrophied as well as dilated. Minor dilatations are also apt to occur under various conditions of strain.

The Lungs and Bronchi.—Chronic congestion and thickening are found in the bronchial, laryngeal, and tracheal mucous membrane. In the lungs themselves the tendency is towards emphysema with atrophy. Bronchitis, broncho-pneumonia, and pneumonia are complications frequently fatal; indeed, recovery from pneumonia is exceptional in alcoholic subjects. The tuberculosis so frequent in them is almost always pulmonary.

The generative organs are sometimes the seat of atrophic and fibrotic changes. The genital function and reproductiveness are depressed, and the offspring of alcoholic parents are specially liable to various forms of nervous delicacy, including idiocy, epilepsy, insanity, and crime.

Among the symptoms of chronic alcoholism there are many that cannot be traced to their anatomical origin, and we are led to infer the existence of changes too subtle to be demonstrated, to explain the *altered metabolism*, the diminished resistance to some poisons and the increased resistance to others. Gout is not directly traceable to alcohol, though its production is largely favoured by rich wines and strong beer. Whilst alcohol intensifies the influence of the gout-producing factors, advanced alcoholism is a form of starvation, and gives little opportunity for the gouty process.

THE CLINICAL USES OF ALCOHOL

ALCOHOL IN HEALTH.—*The psychical, sedative, and narcotic actions of alcohol.*—Alcohol is a luxury rather than a requirement under normal conditions, and a source of weakness rather than of strength for the purposes of sustained work. But in the excessive pressure and wear and tear of modern life it is undeniably useful in easing fatigue, soothing worry, dispelling anxiety, and cheering moods of depression. Its value is that of a psychical stimulant and alterative, but above all of a sedative; and this is also the secret of some of its therapeutic uses. Much less commendable is the resort to alcohol in exhaustion for the purpose of more work. The object may be obtained, but it would have been better secured with the help of less harmful stimulants, such as tea or coffee. Spirits late in the evening are usually a superfluous luxury, and cannot be advocated. Nevertheless, their narcotic effect is often of signal service.

Its Digestive Value.—The dietetic value of alcohol residing less in its nutritive coefficient than in its beneficial effect upon the digestive and nervous functions, it is best taken with meals. Though, like our various condiments, it complicates digestion, a moderation of the wine that pleases favours eupepsia and enables many to digest whose digestion might without it have been a failure. Its place is during or after the meal; only little of it should be drunk at the beginning.

Thanks to the lasting restorative action of food and of sleep, the reactionary depression passes unperceived when the "physiological" daily allowance is divided between the meals and the hour of rest, and the regularity of the habit becomes a protection against any dangerous craving. The practice of drinking alcohol at all times of the day, and especially in the early morning, cannot be too strongly condemned.

Alcohol in Infancy and Childhood.—The well-known effect of maternal alcoholism on fœtal nutrition and development illustrates the pernicious influence which it might exert upon extra-uterine growth, and the exceptional cases in which the alcoholic habit has been established at a tender age in children of the poor entrusted with fetching the liquor from the public-house demonstrate its baneful action in early childhood. During the years of active growth the retarding influence of alcohol upon nutrition and the alcoholic bias are real dangers; abstinence has none. In childhood alcohol is neither required nor missed; the conditions which may call in the adult for its psychical and digestive assistance do not obtain. Although a limited allowance of beer or of wine may be harmless in itself, it should be rendered unnecessary by an adequate diet. Total abstinence until the years of manhood has the recommendation that it is almost protective against the risk of alcoholism in after-life.

ALCOHOL IN DISEASE.—The responsible question, "Should alcohol be prescribed?" arises in every instance of illness, and must be finally decided on the merits of each case, but its solution may be assisted by a study of the indications special to the various clinical groups of diseases of children and of adults, and also of certain broad principles connected with the properties and the mode of action of the drug.

Our guiding principles in prescribing alcohol are based upon its virtues (1) as a stimulant, (2) as a sedative, (3) as a food, (4) as a protection against infection. We may consider these modes of action apart from each other, but in practice they cannot be dissociated.

(1) Alcoholic stimulation, being rapid but temporary, is suited for the prompt relief of remediable conditions, such as physical and mental shock, severe pain, faintness, cardiac failure, etc. Should, however, the affection not be purely momentary but of a more lasting kind, a single dose will not suffice, and we may be led to its continuous administration at regular intervals. This is the practice adopted in pneumonia, hyperpyrexia, malaria, and other acute attacks, and in them we administer it freely and without misgivings, knowing that the attack is likely to be of short duration.

In diseases which run a long course this systematic administration is open to reservations. Frequent repetition tends to lessen the stimulating effect, and if the dose should be increased the nutritive functions may suffer. If alcohol cannot be altogether avoided, it should be prescribed sparingly, and its administration regulated day by day to suit the fluctuating requirements.

(2) Analogous considerations apply to the use of alcohol as *a sedative* and as a narcotic. A single dose may relieve a temporary state of nerve distress, and sleep, if it should be induced, will preclude its being repeated. But when the trouble is lasting or prone to recur, alcohol becomes a complication rather than a help. Its sedative action quickly wears off by repetition, which tends to establish a craving for its stimulating effects.

(3) Though essentially incomplete as a nutriment, alcohol is often in disease the only available support. But it is restorative rather than sustaining, a fuel rather than a food, and not to be relied upon for any prolonged service. Genuine food-stuffs are needed as soon as digestion is re-established, and there is a risk lest alcohol should interfere with a revival of the normal functions of appetite and digestion.

(4) Not least among its virtues is its ill-understood "protective" action. The relief of the shock produced by chemical poisons is due to its stimulating effect; but quite distinct from this agency is the relative immunity which alcohol confers against infection, and the relative protection which it affords against the action of microbes, toxins, and venoms introduced into the system. This protection is probably not due to its neutralising effect upon the poisons, but rather to its influence in raising the resistance or blunting the susceptibility of the nervous mechanisms, or in retarding absorption or checking oxidation. This is an argument for the use of alcohol in the treatment of all toxæmiæ, and may perhaps help to explain the anomalous toleration for alcohol peculiar to the toxæmic state.

As a general conclusion it may be stated that the greatest value is to be obtained from alcohol by reserving rather than by pushing its administration, and by adapting the latter to the requirements as they arise rather than by following a rigid time-table irrespective of the patient's varying needs. To know how to do this effectually is one of the highest developments of the art of nursing.

The indications in diseases of infancy and of early childhood may be considered in connection with four clinical groups.

(1) Clinical emergencies, such as *fits and convulsions*, call for more active measures of treatment than alcohol. As a sedative for the exaggerated

irritability of the nervous system alcohol is inferior to chloroform, chloral, and the bromides, and as a cardio-vascular stimulant it is less needed in infants, whose difficulties are respiratory rather than cardiac, than it is in adults. Nevertheless it may be useful where the stomach persistently rejects food, and when swallowing is impossible it may be injected into the bowel with milk.

(2) Among the acute affections the *exanthemata* do not require its administration, unless they should be of a severe or malignant type; and in all minor ailments it may be dispensed with. Although in childhood *acute pneumonia* usually recovers spontaneously, it has been a growing custom to give alcohol, as in the adult, as a matter of routine. This is usually superfluous, but probably does no harm within the short period covered by the disease. It is the lengthened use of alcohol which we should avoid. In the prostration of severe *broncho-pneumonia* and *whooping-cough*, alcohol may be essential. Diphtheria still calls for its assistance as a prophylactic against depression, though much less urgently since the introduction of the treatment by antitoxin. In acute tuberculosis and in tuberculous meningitis we cannot dispense with its help as a fuel and as a nervine stimulant.

(3) In acute digestive crises, in gastro-enteritis, and in other severe affections it may be indispensable as a restorative and as a temporary supporting diet to tide over a dangerous period. In the virtual starvation of chronic dyspepsia, of enteritis and of rickets among the poor, its help is invaluable, and its timely employment is often the only means of rendering alimentation possible. When digestion is restored, alcohol ceases to be beneficial.

(4) Lastly, it is a valuable dietetic adjunct, even more than a medicine, in profuse suppuration, in anæmia, and in the chronic wasting diseases, such as phthisis, abdominal tuberculosis, and marasmus. In most of these conditions red wine is indicated and usually well borne, whilst brandy is more suitable to the acute conditions and in complete breakdown of the digestion.

The Indications in Diseases of Adults.—In prostrating fevers, when digestion is at a standstill, alcohol is almost the ideal substitute for food, as it needs no digestion and is readily absorbed, largely restraining tissue combustion and a dangerous excess of waste products. In severe pyrexia with delirium considerable quantities of alcohol, which in health would produce stupor, are tolerated, and seem to relieve rather than to oppress the nerve centres. Much of the alcohol may perhaps be burnt off before reaching them, or possibly their nutritional requirement may be considerably raised or their susceptibility to poisons greatly diminished. At any rate, alcohol is thus rendered available as a substantial nutritive supply. The sedative action is displayed in the subsidence of the delirium, and in the lowering of the temperature and of the rate of pulse and respiration. Brandy is the best form of alcohol in these cases, and should be given in small and frequent doses.

In the continued fevers also, and particularly in typhoid, critical emergencies may arise, but, as a rule, digestion is not permanently disabled, and alcohol is neither needed nor desirable as a food. The less of it given the greater the likelihood that appetite may revive for the needful flesh-forming supplies. In uncomplicated typhoid the writer has long given up its use during the pyrexial period, reserving the administration of wine as a dietetic for convalescence.

Often when large quantities are prescribed there is a danger lest, under the veil of delirium, the "typhoid" condition due to the disease may pass almost imperceptibly into an alcoholic toxæmia, which may prove fatal if not recognised—a possibility which calls for the utmost vigilance on the part of the physician.

In pneumonia free stimulation is the almost universal practice in this country, but the absolute failure of alcohol to save life in the worst cases does not warrant any high estimate of its therapeutic value, though it is undoubtedly of use as a restorative and as a food. But it might become a danger were it to disguise from us the often subtle indications for more rational and life-saving remedies.

In acute bronchitis and in severe asthma considerable stimulation may be needed, but the effects of alcohol must be carefully watched, lest it should aggravate the symptoms of carbonic acid poisoning.

In heart disease the uses of alcohol are indicated by the hygienic principles which now govern cardiac therapeutics. If recovery, not euthanasia, is the object of our efforts, over-stimulation is, above all others, the danger to be guarded against. A weak heart needs to be rested, fed, and worked, but not alternately overworked and depressed. Alcohol has its value as a tonic and as a dietetic, but to serve these purposes its allowance must be moderate, and its administration opportunely timed. Oppressive doses of alcohol probably often turn the balance against recovery in advanced cardiac affections.

Diseases of the Nervous System.—In its strongest line of action, that of a nerve sedative and of a narcotic, alcohol is surpassed by other drugs, and it is not resorted to in the worst cases. Neither can it be recommended for its relatively slight antipyretic action. In the treatment of the organic affections of the nervous system alcohol has no place, and in the severe functional disorders of the brain, often partly based upon its abuse, it is useless as a medicine, though sometimes beneficial as a dietetic agent. But during the acute stage of delirium tremens there is less objection to stimulants if they can tempt the patient to take food or remedies.

Among the functional disorders the neuralgiæ are often relieved for a while; but in the worst forms, such as tic douloureux and severe gastralgia, no relief is obtained, and the sufferers are seldom tempted to exceed. The danger of the alcoholic habit is greatest in those minor neuralgiæ and neuroses where alcohol is resorted to for the sake of its temporary stimulation even more than for the relief of pain. It is an invariable rule not to prescribe it in such cases, and to discourage its use.

Renal Diseases.—In acute nephritis alcohol is detrimental. In the chronic forms, including lardaceous disease, it probably does less harm than is generally supposed, and a tonic wine is indicated for the anæmia of chronic Bright's disease.

Acute *cutaneous diseases* are made worse by alcohol, but in chronic atrophic, scrofulous, and degenerative affections every means, including alcohol, must be utilised to raise the general nutrition.

Dyspepsia.—The successful management of a delicate digestion is often a question of the right proportion of whisky or brandy, or of a suitable choice of wine. In prescribing wine for dyspepsia it is even more essential than for other conditions, not to be content with vague directions, such as "Claret" or "Hock," but to specify the kind, and to insist upon quality.

For rheumatic subjects malt liquors are the worst form of stimulant. Whisky, being the least prone to aggravate the tendency, has been credited by the laity with a preventive virtue. In acute rheumatism alcohol is not prescribed except for hyperpyrexia and severe complications. Subacute rheumatism is also best treated without it. In chronic rheumatism the lighter red wines with meals are often indicated, and in old and debilitated subjects additional stimulation may be needed at other times.

In rheumatoid arthritis with debility and anæmia stimulants are wanted and are always beneficial; but in the gouty and in the rheumatic varieties their effects need to be watched and a careful selection has to be made.

The development of *gout* and of its symptoms is favoured by alcohol, which should be avoided in the acute stage. But various complications, chiefly the gastro-intestinal and the cardiac, may from time to time call for it; and in advanced cases, with enfeebled digestion and energy or with cachexia, the need may be a permanent one. Since many who are gouty cannot be prevailed upon to abstain, we should guide their selection of a stimulant. Genuine spirits are probably least harmful, and in prescribing whisky as the exclusive drink we save the patient constant risks from unsuitable wines. All sweet, spirituous, or imperfectly fermented wines, such as champagne, are detrimental. Among the lighter kinds those which act as diuretics on the patient will probably best agree (Burney Yeo). The geographical distribution of gout is a useful guide; it prevails in Burgundy much more than in the Bordeaux district, and it is uncommon in the Rhine country. Light hock or Moselle is thus most likely to suit; claret occupies an intermediate position between Burgundy and hock, and may sometimes be allowed, but in this group the variety is greater and selection more necessary. Similarly gout has always been comparatively rare in the North, and in Scotland and Ireland, where spirit is the popular beverage, and the consumption of ale and stout much less than in the South. The cider-growing districts are also relatively free from gout.

In diabetes the choice is restricted to unsweetened spirits, or absolutely dry wines. Alcohol in some form is generally needed, not only in the acute diabetes of the young, but in the more chronic form of the affection in middle life. Though it may be of little vicarious use in the place of the carbohydrates, it is a stimulating help to digestion. Light clarets which contain little sugar, or the still Rhine and Moselle wines, may be given with advantage for their hæmatinic properties. In gouty glycosuria they may also be allowed; but whisky is more generally suitable, and the diuretic properties of unsweetened gin are less open to objection than in genuine diabetes. In the severe complications of diabetes, such as pneumonia, gangrene, and coma, brandy is the best stimulant and should be administered freely.

In diabetes insipidus, alcohol, even in small quantities, acts, according to Ralfe, like coffee in increasing the diuresis. But whilst avoiding it as a rule we must not forget that the nutritional treatment of the disease is of the first importance.

In all wasting affections, when alimentation fails, alcohol is our last food and support, and even in those visceral diseases which are the result of its abuse, it ceases then to be regarded as a poison.

External uses of alcohol.—Owing to its antiseptic, astringent, and detergent properties, alcohol is of considerable service in various conditions. Thus in strained and swollen joints, muscles, or tendons, and in cases of headache, very considerable relief may be obtained by its use as an evaporating lotion. It may be used alone or in combination with such varieties as vinegar, ammonium chloride, or the like. Alcohol 10 parts, ammon. chloride 2 parts, vinegar 6 parts, and water 60 parts, make a very valuable evaporating lotion. It may be perfumed, if desired, as in the well-known Eau de Cologne.

The hardening properties of alcohol may also be made use of for the prevention of bed-sores (*q.v.*), or for the treatment of tender feet, often accompanied by chilbains. For the latter a small foot-bath—one-third methylated spirits, and two-thirds water—may be very serviceable.

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Alcoholic Insanity.

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Alcoholic insanity is properly regarded as an incident in the development of alcoholism, and not seldom the progressive disease is permanently interrupted by the mental attack. The most dramatic forms of alcoholic insanity are delirium tremens and mania a potu. These are considered in special articles. *Dipsomania,* which is often spoken of as if it were an alcoholic disease, should have the restricted meaning which refers to the constitutional state in which the patient manifests an original and insistent crave for alcoholic stimulation. There are other forms of insanity or of mental perversion which are sometimes described as alcoholic insanity. But in this work we restrict the title to those states which have been induced by alcoholic excess, and which have the features characteristic of alcoholic disease. Alcoholic insanity is a cortical disease in which the lesion which determines the insanity is superimposed upon that of alcoholism. (Just as climacteric or puerperal insanity has a lesion which is added to climacteric or puerperal processes.)

Etiology.—The etiology of alcoholic insanity is very obvious. It is the etiology of insanity plus that of alcoholism. In other words, the disease occurs in persons who are predisposed to the neuroses, and who have drunk to excess. The most usual age for its development is somewhere between twenty-five and thirty-five, when there has been time for the excess of alcohol to have its proper effect, and when the brain is occupied with the complex affairs of mature life. Alcohol specially affects recent cortical structure. After that age the danger continues if the causes persist, and becomes aggravated as the climacteric approaches. A good many cases of senile alcoholic insanity are missed. These are cases in which there may have been no great recent excess, but in which the senile processes reveal an alcoholic injury of long standing. As regards the influence of sex, the truth probably is that female cases are fewer because female drunkards are fewer; but that, given the alcoholism, women are more likely to become insane than men. Not infrequently the attack is precipitated by an acute exciting cause—a domestic, financial, or social crisis, bodily illness, injury to the head, or a great debauch.

Clinical Types.—1. *Cases characterised by a disproportionate impairment of will.*—In the article "Alcoholism" it will be seen how great an importance we must attach to the reduction of the patient's powers of self-control in drunkenness. The physician must regard the inability of the patient to refuse alcohol as a part—and the most important part—of the disease. But at present that is not held to constitute an insanity. The cases in which there occurs an overwhelming desire for alcohol, such as occurs in dipsomania, should be held to manifest an abnormal hypertrophy and perversion of appetite. But the cases to which reference is now made are different. The most usual are those which manifest an insane indolence accompanied with insane impulsiveness.

A man of business, not affluent, had been drinking steadily to excess for years, without showing any great and general mental impairment. Then, unexpectedly, he ceased all work for a fortnight; lay in bed most of the day complaining of ill sorts; rose in the evenings, and often went out in the dark hours. He was not now drinking heavily. At the end of the fortnight he returned to work, but was so careless and unpunctual that he was dismissed. Indifference, however, was still his prevailing state of mind, and even the prospect of poverty did not stir him. One Sunday forenoon, contrary to his habit, he went to church. Towards the end of the service he rose in his place, and with loud voice and in a torrent of language he denounced the clergyman as a Papist. Thereafter he was sent to an asylum. Still he showed little more than extreme loss of interest; he had no gross delusions, suffered no melancholia, was not excited or incoherent, but occasionally cursed, or sang, or threw things about very like an epileptic. There were no convulsions. Tremors were fairly well marked, but no pronounced loss of power was manifest. He complained of tingling and prickling in his lower limbs. The knee-jerks were almost gone. No other positive symptoms of importance were discovered, and the case recovered after a time without further developments.

In a similar case, that of a lady, there was the same indolence, retirement from society, and disregard of social obligations. She lived most of the time in bed or in her back-green. Then she disappeared, and the first news of her was a telegram from Ostend asking for money and for luggage.

In other cases the defect of will takes the form of *Folie du Doute*, an insane dubiety over details, and childish scruples about unimportant trifles. That is usually associated with depression, and again impulsiveness is a feature. In still other cases, while there is no obvious defect of will as regards the common affairs of life, the patient is tormented with insane promptings—ridiculous, extravagant, or dangerous. The patient's fear that some day he will not be able to hold out against these impulses is not ill-grounded. But the worst cases are those in which, without any admission of such promptings, and perhaps without any warning to himself, the patient suddenly plays the public clown, steals, rapes, murders, or commits suicide. This tendency should always be borne in mind in alcoholic cases. Anything may happen until the patient is safely recovered, or demented, or dead.

2. *Cases characterised by conspicuous defect of memory.*—We have a patient now in whom there is no other obvious defect of mind, but who remembers nothing. His is a *defect of retention.* His memory cannot be educated because his brain apparently does not hold any images even for a minute. He plays draughts well, but if you take him from the board and set him down again immediately, having turned the table, he will play his opponent's game and not know that he has changed pieces. He curls well for a beginner, but he can never remember when it is his turn to play, whose side he is on, or which are his stones. He has been for many months in the asylum, but he does not know the name of the house, nor can he with assurance designate a single patient or official in it. As usual in these amnesic insanities, he retains the images of long ago, and can recollect the facts of his boyhood. But he now has no consecutive consciousness. Much more common is a *defect of recollection.* The patient's

brain retains images, but he cannot "lay his hand" on them. This disability is most pronounced as a form of aphasia. Many alcoholics can hardly finish a single sentence correctly, but, if they stick, they are aware that the word has just evaded them for the moment, and if they use a wrong word they may recognise that it is wrong. This defect of recollection may refer to special things. Many patients, for example, are specially unable to remember time. They do not recall meals, nights, mornings, and other landmarks which we unconsciously use to record the passing of hours and days. Other patients can remember what has gone, but cannot remember what is coming, or *vice versa.* Others have specialised forgetfulness of more curious sorts. The most grotesque mistakes depend upon a *defect of recognition.* The brain stores its images, recollection or representation of them is active, but mistakes occur when a judgment is asked which would confirm or deny identity. Thus many patients use the wrong words with complete assurance, or have a sense of recognition of new surroundings which ought to occur only in familiar circumstances. Mistakes as to the identity of people are very common. Some alcoholic patients will accept any evidence or suggestion as to who a man or woman is, and utterly fail of the corrective judgment which their senses ought to supply. It is an interesting fact that such patients remember the personality of an acquaintance when they forget his appearance, and will invest the usurper with all the spiritual qualities of the person whom he impersonates, although there may be no obvious outward resemblance whatever. In my opinion we have here again a convulsive or fulminating psychosis—a discharge in certain mechanisms which is automatic or nearly so. Assurance normally occurs to clinch a consecutive process of comparison when, by association, we reach given mechanisms, and discharge them with a result which is known in consciousness as certainty. But alcoholics have all the pleasant certainty of assurance at the wrong time or in a wrong connection, which betokens the instability of their mechanisms. A very curious and interesting defect—most common in senile cases—is an *excess of reminiscence.* When recent images have faded and are lost, old images, the memories of early life, come vividly into consciousness. Some old alcoholics will regale their audience by the hour with revived memories, and furnish them forth with a graphic circumstance and detailed realism which suggests that they imagine as much as they remember. This inability to discriminate between what might have been and what was, characterises the conversation of many alcoholics in a lesser degree. Some patients who are not greatly at fault in general, seem to have very little sense of probability in reminiscence.

3. *Cases characterised by disproportionate defect of judgment.*—Almost no one suffering from alcoholic insanity escapes delusions. That sense of probability, the abeyance of corrective judgment, which has been referred to in operations of the memory, may also be evident when actual and present facts are offered to the mind. In a great many cases the delusion or delusions are the most obvious fact in the disease. I have a patient now, a case of alcoholic insanity, who has the most elaborate and systematised delusion of persecution I have known. Most commonly the patient is haunted by some kind of fear, and having once realised that, he deduces *ad libitum* and without almost any discrimination. Everything supports his premise. According to temperament, habit, or circumstance, the alcoholic fear may be interpreted in various ways. The most common delusions are—that attacks are being made upon the person by a fell disease, by spirits, by electricity, or by a conspiracy of people; that God has been mortally offended, and will punish eternally; that financial ruin is inevitable; that every one loathes

and shuns the patient; or, more vaguely, that an unknown horror is impending. On the other hand, not a few cases exhibit exalted delusions. Many alcoholic cases greatly resemble general paralysis in the grandeur of their delusions, and others, without great extravagance, believe themselves to be specially worthy of praise and distinction. Many of the alcoholic expressions of innocence are as much delusion as deceit. The patient still believes in himself as a moral person, just as he still believes in his power to do well. Most important are delusions which have a basis in hallucination. The most grave thing about hallucinations of hearing, for example, is that the patient not only believes that these sounds have an objective occasion, but that he builds upon them a huge fabric of nonsense. That is the case with our patient last referred to. He has a complete and very complex system of false beliefs about a conspiracy of evil spirits who persecute him. He has, as has been commonly observed since Magnan described it, many perversions of common sensibility and many painful paræsthesias, and these he attributes to devilish operations of his enemies. This is confirmed by the conversations which he holds with them—angry and long and loud. He has taught his hallucinations to answer him, to hold coherent conversation with him, though, perhaps, as many dialecticians affirm, the remarks of the opposite party partake more of invective than of argument. When a patient gets that length—and it is not uncommon—he has most convincing reasons for his delusion. So deep and extensive an organisation of errors of judgment almost precludes the possibility of recovery. Fortunately many of those most systematised delusions accompany a depth of impairment which obviates violence. For it must be remembered here again that impulsive violence characterises a great many delusional cases of alcoholic insanity. In the early stages of a delusion especially accidents must be looked for. A ruined man, a man eternally lost, a man over whose head some horrid sword is hanging, is extremely likely to put himself out of suspense and misery. And people who are persecuted, who are the victims of conspiracy, are very apt to turn. When that happens they may wreak their wrath with but little discrimination. For nearly all such delusions attach to some innocently offending person who is in the immediate neighbourhood, and who, in the patient's regard, is an accomplice or an enemy in disguise.

4. *Cases characterised by melancholia.*—Alcoholism accounts for a large number of the melancholics in our asylums, and the type of insanity from which they suffer is characteristic. It is a melancholia which is accompanied by some of the symptoms which we have considered. A simple melancholia practically does not occur in alcoholics. Delusional melancholia is very common, and the delusions are such as we have noted. There is in nearly every case an impairment of memory which is greater than in simple melancholia; and there are the defects of control which is usual in alcoholics. In particular, alcoholic melancholia is often attended by impulsive violence. Every alcoholic melancholic must be expected to make an attempt upon his own life or upon his neighbour's. And such acts are more to be dreaded in the earlier stages when the melancholia is developing. Men are more apt to become melancholic; women, maniacal.

5. *Cases characterised by mania.*—The immediate form of mania, which we call *mania a potu*, is described in a special article. It may develop in a case which does not manifest many of the symptoms of alcoholism. Most of the alcoholic manias pass through a stage of melancholia. Their excitement is very often of the angry type, especially when accompanied by hallucinations. But more frequently the type of mania is exalted or

jubilant, if not quite hilarious. In women especially a mania is common which is more delirious—which does not take any notice of surroundings. In both sexes, but chiefly in men, there is great difficulty often in diagnosing between alcoholic mania and excited general paralysis. Time alone can make us certain, because not a few cases of alcoholic insanity do incur general paralysis. In many cases we must take note of the fulminating, eclampsic nature of the excitement. At one hour the patient may be reasonable and calm, apparently on the high road to recovery; and the next wildly excited and violent, without any apparent cause, and without much of a transition stage. In some cases there is an approach to an aura. Some patients know that an attack of short mania is imminent.

6. *Cases characterised by partial but permanent dementia.*—A quite considerable number of insane alcoholic cases manifest a dementia which is not sufficiently recognised. A fair proportion of the sensational cures of which one hears prove to be "cured" by dementia. The patient passes through a prolonged stage of drunkenness, through an acute stage of nervousness in some form, and then comes into the world again a sober and a stupid person. But the same stupidity—the irrelevant talk, the platitude of mind, the lack of perception, the dulness, the want of initiative—may occur without an acute attack of any kind. While the patient is taking an excess of alcohol his mental functions are active though perhaps perverted. But if the alcohol be withdrawn the mind proves itself disabled in all its relations, and continues to be so. The occasion of the appearance of the symptoms is the cessation of alcohol. The peculiarity of the dementia is that it is not progressive, but that also it does not resolve. When all alcohol has been eliminated, when the bodily functions are apparently quite sound and good, still the mind does not reawaken. Such a case, in a young man or woman, will probably pass into a phase of acute illness at the climacteric or in senility. A progressive dementia, proceeding to an almost complete mindlessness, is the typical course of alcoholism, and, in its later stages, it is an insanity—an acquired state like the congenital defect, imbecility. But, as has been remarked, an acute attack of insanity, leading to enforced sobriety, may permanently interrupt the dementia.

Pathology.—Discounting cases of gross lesions which often occur in alcoholism, and which may occasion an insanity, there is little to be said as to the pathology of this disease which will not be found in the articles "Alcoholism" and "Insanity." However we explain it, one of the facts of the insane diathesis is that the cells and fibres of the cortex cerebri are not of normal stability. That refers not only to their readiness to discharge too quickly or too deeply; it refers also to their defect of resistance to harmful conditions. High temperatures, fatigue, toxic agents, and other ills tell upon these sensitive mechanisms more than upon those of a stable brain. The disintegration of structure which occurs in nearly all alcoholics in some one or other realm of the nervous system is accordingly apt to be deeper and more extensive in the cortex of a drunkard who is of neurotic habit. What the histology of such a process is believed to be may be gathered from the articles referred to.

Treatment.—Similarly, the treatment of alcoholic insanity is simply the treatment of insanity plus the treatment of alcoholism. But it seems advisable to repeat our reference to the impulsiveness of alcoholic insanities, and to suggest that, having regard to it, no time should be lost in removing the patient to a place of safety, and that, meanwhile, supervision should be effective and absolutely complete.

LITERATURE.—1. Art. "Alcoholism."—2. Hack Tuke. *Dict. of Psychol. Med.*

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DEFINITION.—Alcoholism is a disease of the nervous system due to alcoholic excess. It is essentially and always a disease which attacks the central nervous mechanisms, and, in its normal course, it is a dissolution or progressive reduction of function. The highest, that is, the most voluntary, cerebral functions suffer first, and are most deeply impaired. But there are, in most cases, impairments of motion and of sensation, and derangements of the trophic function of nerves; and in some cases there are affections of the peripheral nerves and ganglia, and visceral complications. These low-level impairments, however, are always secondary to the highest central disorder. That disorder is manifest in a purposive (volitional) defect, which relates in particular to the appetite for alcoholic indulgence, but which becomes manifest also in relation to conduct in general. A proper account of alcoholism is therefore a description of the appearance of a defect of will in relation to alcohol, and later, of various developments which are common to the disease in one or other of its stages. Its earliest stage is popularly known as drunkenness.

DRUNKENNESS.—Drunkenness develops in an individual in this country along fairly constant lines. There is no hard and fast distinction between drunkenness which is vice, and drunkenness which is disease, or, as physicians might express it, between physiological and pathological alcoholism. There are many who hold that alcoholism is never physiological but always unhealthy; and in their view all drinking means some degree of disease. But we must come to a clear understanding of when a patient has arrived at that stage to which the term "alcoholism" in its technical sense can be fairly applied. The general practitioner will often be called upon to say whether his patient really is suffering from alcoholic disease, and he must have a constant criterion which can be understood of all, and which is easy of application to every case. To that end it is needful to conceive of alcoholism always as an affection which primarily involves the will. The fact that the man or woman has become a "patient," that a physician's opinion has been sought, entitles the practitioner to form a judgment concerning the drunkard's habit, even although the patient complains only of some remote symptom such as neuralgia or alcoholic morning sickness. If such a patient can resist alcohol and refuse it, he does not suffer from alcoholism in the medical sense. If he cannot abstain for such time as the physician prescribes, he does suffer from alcoholism. That merely corroborates, with a scientific implication, the popular verdict that a person is a drunkard when he cannot remain sober. The vulgar criterion of sobriety, however, is not sufficient for our purpose, because many patients suffer from alcoholism who have not been often what is generally recognised as "drunk." To understand such a development we must consider somewhat fully the stage which we may call the Inception of Alcoholism—the facts of the development of the alcoholic vice on its way to becoming a disease.

INTOXICATION.—Intoxication in the popular sense is quite often an abnormal effect of alcohol. As physicians we must recognise as intoxication a state which often escapes the attention of the uninitiated. After the first stage of general exhilaration the normal effect of alcohol upon the nervous system is in the nature of an obliteration of the functions which constitute self-direction, and which imply a full consciousness. By degrees the person becomes less aware of everything—of his surroundings and of himself. He may realise this as a drowsiness which is creeping over him, and he may abstain. If he continues to drink he will become comatose, or if he has drunk a great deal before he falls asleep. So regular and gradual a narcosis is most apt to occur in persons of very stable organisation and generally under the influence of a beverage in which the alcohol is pure, well diluted, and not too rapidly imbibed. But it is easy to understand that a person who can become so unobtrusively intoxicated, who drinks himself slowly to sleep, who does not become in any sense loud, and who perhaps postpones his sleep until he reaches his bed—such an one may never be suspected of drinking greatly to excess. Yet he is quite apt to become the victim of alcoholism.

Anomalous intoxication is much more recognisable. As regards cerebral symptoms there are two important effects, each of which characterises a class of intoxication—stimulation, with excitement or excess of activity, and narcosis, with paresis or diminution of activity. Either stimulation or narcosis may be excessive or irregular in the sense that they invade one or more functions disproportionately; and such anomalies of intoxication are important because they typify anomalies of alcoholism. For example, the narcosis may be disproportionate in the motor centres, so that a man may "keep his head, but lose his legs," his speech, his fingers, his eyes. There may be similarly an excess of sensory paresis, an anæsthesia of the lips or fingers, or a dulness of hearing. In another class are the anomalies of intoxication which are characterised by symptoms of excess. The most obvious type is the "drunk and disorderly" person who shouts, or dances, or is violently destructive long after he has "lost his head"—an excess of activity in motor centres which is disproportionate to the general narcosis. Even in a semi-comatose state, when the upright posture is impossible, there may be persistent local excesses, either of motor or sensory functions. Both of these kinds of anomalous intoxication are easy of detection. It is the fortune or the misfortune of some persons that they become obviously drunk long before narcosis is complete, before they become drowsy. They also are apt to become victims of alcoholism, though not more so than those who become inobtrusively drunk. Their alcoholism, however, is likely to be anomalous.

More unusual are those in whom the visceral effect of alcohol is very great, in whom, for example, nausea or palpitation supervenes, while they are still in full enjoyment of sense and of motion, or in whom the derangement of liver next day is out of all proportion to the purely nervous reaction. Some of these cases are perhaps truthfully described as instances of a peripheral nervous effect. They also typify forms of alcoholism—in which visceral disorders are out of proportion to the central impairment.

By many acts of intoxication—regular or irregular—the patient arrives, often insensibly, at a stage in which his central nervous system has incurred a disease characteristic of his habit. The effects of intoxication depend upon the personal equation, the nature of the drink, and the mode of drinking. Normally, the case develops into that state which is called Chronic Alcoholism. But on his way to it the patient may suffer one of several developments incidental to the disease.

INCIDENTS OR INTERRUPTIONS IN ALCOHOLISM.—The affections here discussed are all recognised as separate diseases. They commonly occur in the course of general alcoholism, but any one of them may be an acute development in a patient in whom the general disorder is not very far advanced.

ACUTE ALCOHOLISM (*Alcoholic Poisoning, Alcoholic Coma*).—Acute Alcoholism occurs most commonly as the sequel of a silly wager made by some boy or man that he will drink so many bottles of beer or glasses of spirit in an absurdly short time. But it may also occur as a consequence of the removal of forethought and prudence under the influence of alcohol. I am convinced that this is one form of anomalous intoxication; and it has not received sufficient attention. In normal cases the taste for drink subsides under a general narcosis. In other cases the person still appears to desire drink, at least he swallows it with some semblance of enjoyment, long after he can enjoy most other things. That is a very dangerous kind of effect, and any one who experiences it should be put upon his guard. (Memory should be the criterion. It is dangerous to drink more than one can remember.)

The symptoms of Acute Alcoholism are those of Coma, and need not be discussed. Diagnosis, however, may be difficult, and should always be arrived at with great care. It should be a strict rule that all persons found comatose on the street should be taken to hospital or examined by a doctor before being consigned to the prison cell. The states which ought to be excluded in diagnosis are—Head Injury, Apoplexy or Embolism, Uræmic or Diabetic Coma, and Narcosis from some drug other than alcohol, especially opium or morphia. The real difficulty is that the case is often complicated, the drinking having preceded one of the above states. In pure cases diagnosis is generally easy. The following are distinctive features. The pulse is often full and bounding, while the respirations are deep and slow; the pupils are dilated; the temperature is subnormal and the same on both sides; muscular twitchings may occur, convulsions rarely, and reaction to stimulation may be evoked in the various muscular departments of the arm or leg, or the patient may be incited to speech. Treatment is practically the same as for any other narcosis. The stomach should be washed out. Emesis is neither so safe nor so satisfactory. I have repeatedly seen alarming collapse follow apomorphia. Careful washing out need not put any great strain upon the heart, especially when the patient is too comatose to struggle. The operation should be performed slowly. Thereafter, administer a purge and induce diuresis and diaphoresis. Give a large enema if there is no collapse. Then put the patient to sleep in a large room with plenty of fresh air. The rousing treatment usually practised in opium narcosis is not indicated, but the patient should be aroused if the respirations become very slow. In all narcosis sleep is useful, even if it has to be very frequently interrupted. Some cases manifest shock, which must be diagnosed and treated as distinct from acute narcosis.

MANIA A POTU (*Immediate Maniacal Alcoholism*), ALCOHOLIC INSANITY, AND DELIRIUM TREMENS (*Delirious Alcoholism*) often occur at a comparatively early stage of alcoholism. Each of them, however, implies a greater or less degree of general alcoholic disorder. They are treated under the above names in their alphabetical place in this work. It is sufficient to say here that these affections constitute disproportionate impairments of certain nervous mechanisms, in such a degree as to conceal the general alcoholism, and in each of them an interruption of the general disease is implied. In ALCOHOLIC INSANITY disorders of the attention are in excess—some kind of impairment of the psychical functions of the brain. In MANIA A POTU the disease is deeper. Attention is perverted, and then is added an acute disturbance of excess in the mechanisms of purposive (voluntary) movements. In DELIRIUM TREMENS there is a disturbance of excess on a still lower level—in mechanisms which have not a great voluntary relation—which is evidenced by fever, tremors, and great sensory disturbance; while there is also a perverted attention.

CHRONIC ALCOHOLISM (*Alcoholism Proper*).—If the excessive drinker survive these various incidents in the career of drunkenness, or if, as is more usual, he escapes them, he will by degrees develop symptoms of a gradual dissolution or reduction of nervous function analogous to the progressive disability of intoxication. Enough has been said to suggest that a Typical Alcoholism is rare: in fact it does not occur, because alcoholic drinks are not constant, and because human brains are not typical. Apart from those acute states which have been noted and which are to be regarded as mere incidents, alcoholism is very manifold. Its varied nature, in so far as it depends upon the personal equation, need not be considered at length. Granted that alcoholism is a reduction of faculty, it is to be expected that these impairments will occur irregularly, because no two people develop alike. There is one kind of mental defect which is quite constant, however, although patients manifest it variably—the defect of volition, to which reference must so insistently be made. The next most common effects are as regards memory and as regards belief; and, less commonly, the feeling of the patient may be altered so that he is either exalted or depressed in moods which have no reference to his circumstances. These mental reductions, it will be noted, are such as characterise the chief forms of Alcoholic Insanity. We only call such states insanity when they are disproportionate in degree and excessive in duration. In a usual course, for example, delusions do not persist in an alcoholic, because of a progressive dementia which prevents his thinking out a delusion. The same progressiveness characterises the invasion of all the mental functions by alcoholic disease. Excess of activity in the lower functions of intelligence gives place to a gradual impairment of them, and a disproportionate defect of any one of them, such as occurs in the amnesic form of Alcoholic Insanity, becomes lost in a total defect of mind. And so in a further stage of Chronic Alcoholism the reduction of mental function is seen to be very considerable and very general. The patient then has almost no will, no desire, not even an initiative towards drink; no emotion or sentiment, not even regret, much less any hope; no apparent memory, no expressed thought. By this time there is a very obvious impairment of the lower realms also. Following the inverse order of evolution the reduction is first manifest in the most highly differentiated co-ordinations. For example, skilful manipulations of all kinds are impaired, and if the patient be a technical artist his art will obviously suffer in technique as well as in idea. The incoördination in nearly every case will become manifest in an impairment of such fine movements as handwriting, shaving, buttoning, and refined articulation. At the same time, impairments in delicacy of sensation occur, but these are more difficult of detection. At all events, it is certain that there is no evidence of refined sensations, no sign that the patient notices refinements of sound, of colour and form, of taste, smell, touch and temperature. But it is not easy to say whether he is absolutely not aware of these things, or whether he only cannot respond to them appropriately. While this expressional and sensational disability is progressing, secondary symptoms occur, of which, on the one hand, tremors are the most notable, so marked sometimes as to greatly simulate General Paralysis, and, on the other hand, paræsthesias, which become illusions, and those apparently automatic activities which we call hallucinations. But these also disappear in time as the lesion deepens.

Still later the patient is yet more deeply paralysed. Not only has he lost refinement of co-ordination, but there is an actual loss of muscular strength and a disability even in gross movements. At this stage the muscles generally are flaccid, as, for example, in the face, which is flat and expressionless. The hands and arms are flabby, the grasp weak, the step devoid of spring. Fatigue is imminent, the carriage is slack, the patient stoops and totters. Now also the impairment of sensation is very evident. Even extreme stimulations are not appreciated. By this time also the deeper and more vital functions are involved. The pulse is soft and irritable and precarious, the extremities cold, sometimes cyanotic, respiration shallow, intestinal peristalsis feeble; and the organic and deep reflexes are impaired, so that one finds irregular reactions in the pupils, the palate, the knee-jerks, and the skin reflexes, and very often the patient has much difficulty in swallowing and may have incontinence of urine and of fæces.

So great a reduction generally implies a cessation of drinking, and the end usually comes by intermittent disease. But if any one were brutal enough to pour alcohol into such a dement the last stages of the dissolution might be reached. As a matter of fact the last possibility of mental reduction seems to be reached in cases which actually do occur. Without accessory disease, however, patients do not, except very rarely, arrive at the penultimate stage when, with great trophic impairment, there is general motor and sensory paralysis. At the last the alcoholised patient, mindless, motionless, senseless, would cease from even vital activities by an atrophic dissolution of the mechanisms of respiration, in which other essential centres would also be involved.

VARIATIONS OF ALCOHOLISM.—A Typical Alcoholism, as above described, cannot be said to occur. There are, in every case, great departures from the type of a general consecutive paralysis. These variations constitute, as in intoxication, two kinds of anomalous disorder. One is a disproportionate local paralysis—a dissolution of a function or group of functions which is in advance of the general dissolution. The other is a disorder of excess—a survival of activity in a function or group of functions which is out of keeping with the general dissolution. It would be unsatisfactory, however, to arrange the anomalies of alcoholism under these two headings, and tedious to refer repeatedly to them. I therefore leave the reader to decide, as regards each symptom mentioned, whether it falls into one class, or into the other. An illustration of each will suffice. Aphasia which is pronounced, which occurs in a patient whose general memory is fairly good, who can read and write words readily, etc., though he cannot speak them—such an aphasia is a disproportionate and anomalous dissolution. On the other hand, a patient may have a very disordered general memory; he may be very stupid, he may have epileptic fits, tremors in his limbs and face, incontinence of urine, etc., and yet he may gabble almost constantly, and with only a very slight defect of articulation—such an excess of conversation is a disproportionate or anomalous survival of function.

I have already alluded to the arrangement here followed. Some of the symptoms now to be alluded to may occur in such a degree as to characterise the alcoholism so that one would almost be justified in giving a special name to one or two of these variations. Most of them, however, occur along with a great general dissolution, and are conspicuous only for a short time. Most of them occur at one stage or another in nearly every case which is not interrupted. It is only when they persist anomalously, or when they occur prematurely, that we are justified in speaking of them as variations. We may take it, then, that these are the symptoms frequently observed in alcoholism. But the disease is so manifold and so general that it is impossible to enumerate all the facts; and accordingly I select what seem to be the most important.

PSYCHICAL VARIATIONS IN ALCOHOLISM.—The most important psychical anomalies are discussed in other articles (see "Alcoholic Insanity," "Mania a Potu," "Delirium Tremens," "Insanity," "Dipsomania"). In the article "Alcoholic Insanity" the psychical facts are discussed at some length. Any of the symptoms there alluded to may occur in some disproportion, though perhaps not to such a degree as will brand the case as one of the characteristic insane types.

MOTOR VARIATIONS IN ALCOHOLISM.—Tremors and spasms are the most usual motor anomalies in alcoholism. They both occur most commonly in the most voluntary muscles—voice, tongue, lips, face, hand—but they may be general, or they may very rarely be most marked in the muscles of the lower limbs. As a rule they are aggravated by attention, as in Chorea and in Delirium Tremens. The tremors are to be distinguished, as a matter of scientific insight, from the spasms; but in practice even authorities describe muscular states without any such distinction. In the tongue, for example, one may observe a fibrillar agitation which is really a fibrillar spasm; a fascicular contraction which gives the vermicular appearance (as if a worm were wriggling in the muscle substance); a choreiform jerking of the tongue out or in, or to one or other side; or a cramp, a tonic spasm, of one or other of the groups of fibres, in or below the tongue. But there may be tremors also with or without the spasms, in the sense that there is a want of balance between opposing muscular fibres, so that the tongue shakes sideways or to and fro. Similar phenomena are present in the hands. There may be spasm, fibrillar, fascicular, or in a muscle or group of muscles; or there may be a tremor of a small kind, or so great as, for instance, to upset a glass of water and restore it again to its upright position. In the jaw spasms are very common, and cramp in the muscles of the lower limbs is quite frequent; of all such excesses epilepsy is the most impressive, though not by any means frequent in alcoholism; unless, as is not improbable, *petit mal* is much more frequent than patients admit. Alcoholic epilepsy may be general and resemble true epilepsy, or there may be a local tonic spasm, followed by clonus, that is, an epileptiform seizure. Alcoholic fits, as a rule, are successfully treated by abstinence, and by improving the general health. That fact in therapeutics should be regarded as a symptom also, revealing the nature of the lesion. Persistent convulsions may be diagnosed as non-alcoholic, that is, as due to a lesion which is perhaps added to an alcoholic lesion. [See "Epilepsy."]

Motor paralysis of almost any kind may occur in alcoholism. We must, however, discount such a paralysis as follows a gross lesion. That is a complication of the alcoholic disease, not an essential part of it. It would probably be considered rash were we to admit that, for example, hemiplegia might occur as due to the microscopic lesion which I take to be the lesion of alcoholism; but there are cases when rapid recovery under abstinence suggests that this may be so. If aphasia occurs in alcoholism without a gross lesion, why not hemiplegia? Local pareses, certainly central in origin, do occur. The difficulty is to discern what is absurdly called the hysterical or functional element—which is really a matter for psychology. An aphonia for example, which is not uncommon in alcoholism, a temporary loss of the use of a limb, or a temporary paralysis which the patient assumes to be complete (believing himself unable to move), certainly implies a lesion which affects the patient purposively, so that he does not try his best to speak or to move; but such a state of mind is almost certainly induced by a state of motor centres or nerves, in which the patient has really tried to exercise the muscles and has failed. That the delusion persists after the temporary motor paralysis has disappeared does not diminish the gravity of the motor disability. That a paresis of phonation does occur, there can be no doubt. Quite commonly also there is a marked defect of speech. Aphasia has been referred to as part of Amnesic Alcoholic Insanity. But a disability in articulation, which is usually associated with marked tremors and spasms, is very common, and it is often aggravated by effort, so that the patient does really find it impossible to make his tongue and lips complete a sentence. The paresis, on the other hand, may become conspicuous in much less voluntary movements. Deglutition may be impaired, or there may be interference with the vesical or rectal reflexes, out of due time. Paraplegia sometimes occurs; but that, like Neuritic Paralysis, may perhaps be best regarded as due to a new and distant lesion.

SENSORY.—Psychical impairments in Alcoholism are usually obvious, motor impairments are almost necessarily so, but sensory defects are often concealed. In all probability there is, in every grave alcoholic case, a quite considerable paresis of sensation. But while the patient is ill he does not refer to it, and if he recovers he has forgotten it. Muscular and tactual sensations may be observed to fail, or their failure may be implied in the disability which prevents graduated refinements of effort, but neither that nor a failure of any of the special senses is usually prominent or complained of in alcoholic disease. Much more commonly the disturbance does not apparently get beyond a lesion of excess. Paræsthesia of all sorts is common, illusion and hallucination. Of these, hallucinations of hearing are the most impressive. They may be both elaborate and persistent. Paræsthesia of hearing—an illusion—is probably commoner than is supposed. Any kind of auditory stimulation may be mistranslated into a wrong auditory perception. That is certainly the case with vision. Hallucinations of sight are not very common, though they do occur. As a rule, they are simple and not elaborate—clouds, lights, colours, flashes, etc. But quite commonly the patient suffers from visual illusion (paræsthesia not discerned), perceiving people or beasts in foliage, trees as men, or significant forms in fire, smoke, or cloud. It is impossible to say how much of the symptoms is due to a real paræsthesia, and how much to a disordered judgment and perverted imagination. The same difficulty arises in estimating the perversions of taste and of smell which are common in alcoholism. Similarly, the "hallucinations of skin" may develop out of real paræsthesia. A sense of heat or of cold, of a localised blushing or blanching, an itch, a cold trickle, a crawling, a sense of small movement (as of a worm) under the skin — these may be modified, minimised, or amplified, according to the vagaries of the patient's attention. In estimating pain especially there is great difficulty. No doubt neuralgic symptoms are very common, but it is impossible to get at the truth about them. For the same reason anæsthesias of all sorts can only be fallaciously investigated. One meets with cases of complete, though transient, loss of one or other of the special senses. Local anæsthesias of the skin and misreference of slight sensation are very common. But in nearly all such cases, when the lesion has got the length of grave sensory impairments, one is dealing with a person in whom observation of self is notoriously misleading, and whose attention is very precarious.

VISCERAL, VASCULAR, AND TROPHIC VARIATIONS.—I now speak, not of such lesions as primary structural alteration in the blood-vessels or other organs, but only of neuroses.

Of the visceral-neurotic anomalies in alcoholism by far the most important, in my opinion, are cardiac. Hurried heart and other disturbances of rhythm are very common, and disturbances of cardiac force. The thumping action of the heart may be very distressing, and cardiac failure, sometimes amounting to syncope, is even more common. These may or may not be associated with palpitation and with angina. Very common also are vaso-motor disturbances—especially such localised congestions as occur in the skin, and which probably also occur in the viscera, and perhaps in the brain or meninges. Asthmatic conditions and hiccough are common. With regard to other viscera, the general rule applies that nervous disturbances in alcoholism are in proportion to the higher relations of visceral mechanisms. In this connection it is suggestive to note the frequency of variations in the lachrymal and in the reproductive functions. In all probability the Vagus nerve and the Solar Plexus are the vehicles of disturbance in many of the functional disorders of the thoracic and abdominal viscera. Affections of the sympathetic nervous system are of peculiar interest in alcoholism, and have not been deeply studied. Much less problematical are the peripheral and muscular impairments which are commonly understood to signify an interference with the trophic function of nerves. Such a lesion has usually a definite relation to vascular conditions, as when pigmentation occurs. The tone of voluntary muscles is admittedly impaired in alcoholism. On a par with that is the flabby heart of many alcoholics and the atonic bowel. But, again, as in considering the liability to fatty and cirrhotic degenerations, it is impossible to appreciate the relative importance of an impaired trophic function and of an alcoholised blood. It seems not unreasonable to suppose that some visceral idiosyncrasies in alcoholism—cases in which non-nervous symptoms are conspicuous—are predisposed to by a neurosis of visceral mechanisms, though the subsequent disorder is not a neurosis. These are here regarded as complications.

It is impossible to conceive of neuro-muscular relations without the implication that muscles depend upon energy of central origin for their dynamical capacity, and when, as in alcoholism, the central system is depressed, the muscle fibres are correspondingly depressed. A similar depression may occur in visceral, cutaneous, and other structures. But, as has been said, it is easy to exaggerate the importance of such an effect, and to depreciate the effect of alcoholised blood. Whatever the explanation, the important fact remains that healing processses are impaired in alcoholism, and all the tissues devitalised.

COMPLICATIONS OF ALCOHOLISM.—What has just been said prepares for the remark that *the alcoholic is abnormally prone to nearly all diseases,*—that almost any other disease may complicate alcoholism. The most ordinary complications are digestive; the more grave are cardio-vascular, pulmonary, and nervous. Of these grave affections the more important are in the cardio-vascular system—fatty heart, cerebral hæmorrhage, and thrombosis; in the respiratory system, pneumonia, phthisis, and bronchitis; and in the nervous, neuritis, hysteria, myelitis, and meningitis. The morbid changes which characterise these and other conditions are considered elsewhere, as they are really diseases in their own right, so to speak, and not merely accessory to the dissolution of the nervous system. Cirrhosis of the liver, for example, which is perhaps the most distinctive alcoholic affection of non-nervous tissue, is a degeneration which may have almost no direct relation with the lesion of alcoholism proper.

PATHOLOGY.—It must be admitted that up to the present time we are not in possession of a histology of alcoholism which squares with the clinical facts, although we are probably on the verge of such a pathology. We must, accordingly, keep clearly in mind the phenomena of which a pathological explanation is required, rather than commit ourselves to a conception of the morbid changes based upon results which every one admits to be far from complete.

At the outset we have to bear in mind that the primary fact about alcoholism is the impaired excitability of nerve mechanisms which characterises it. That is not in contrast with the increased instability of lower and surviving mechanisms. We must have regard to abolition of function both in intoxication and in chronic alcoholism. Moreover, the morbid stability of the cortical mechanisms is often of a systematic nature —that is, it partakes of a consecutive paralysis, which implies a distribution of the lesion in functional, and not merely in adjacent, areas. But, though the impairment often follows a course which suggests that the lesion is progressing along lines of function, there are undoubtedly symptoms which indicate also a contagious spread of diseased processes.

Essentially the lesion of alcoholism is, in my opinion, an *affection of the nerve elements* in the cortex, but that must only be accepted as an individual opinion. With such a premise we must immediately observe that vascular changes, especially in the higher centres, are most intimately related to the changes in the nerve-cell and its processes, and that the two go hand in glove to occasion alcoholic dissolution. It has previously been stated that gross vascular lesions are to be regarded as complications, rather than essentials, of alcoholism. That statement may now be modified so as to make it apply to apoplexy, embolism, local congestion, etc., which is part of a general arteritis and associated possibly with Bright's disease. But undoubtedly a great number of alcoholics escape disease of the larger vessels. On the other hand, disease of the small cortical arterioles must be regarded as essential, because of the very intimate relation which must always be assumed between a cortical mechanism and its vascular supply. A group, or constellation, or tract of nerve-cells and fibres can never be supposed to incur disease without a coincident affection of some nature in the vessels which supply and are controlled by it. Connective tissue changes, on the other hand, are, in my opinion, always more remote. They represent phagocytic processes, that is, part of the mechanism for the elimination of waste matter, and later a substitution process whereby the lower forms of tissue abound when the failure of nutritive conditions has made the persistence of higher tissues impossible.

When we turn to histology for an explanation of the diminished and perverted reactions of alcoholism we must, in the first instance, have regard to the nerve-cell and its processes. In the elements, especially of the deep cell-layers of the convolutions, changes occur both in the non-conductor parts of the cell and in the fibrillar portions which the nerve-currents traverse. These are as yet indiscriminate, but the following facts have been noted. In the non-conductive portions of the cell the chromatic bodies may be displaced from their perinuclear position towards the periphery, scattered variously in the cell-body, or pulverised. The displaced and fading nucleus, the swollen and distorted cell-body, the hardening of peripheral protoplasm so as to develop for the first time a cell-wall, the aggregation of pigment granules under it, truncation of the process cones, fatty globules, and vacuolation, precede the complete disappearance of the cell. The changes in the conductive portions of the cell—the fibrillæ of the processes and of the perinuclear filar network (cytoreticulum)—have not been made so clear. Theoretically, however, the changes in them are of the first importance. The lesion, whatever it may appear under the microscope, is in the nature of a trophic defect which spreads as an ascending or descending lesion to functionally related mechanisms. For it must be remembered that the nerve-mechanisms of the cortex are as dependent upon their relations for their trophic supply as are the nerves of the lower realms, although the channels of it are much more diffuse. There seems some reason, however, for the hypothesis that structural changes do not initiate in the fibrillar portions of the cell, but that conduction is impaired as the result of the changes in the chromatic portion, and that by the time the fibrillæ become so structurally altered that we can observe the condition the lesion is necessarily very grave and implies the atrophy and disappearance of the whole cell (neuron). Certain it is that complete wasting of the cells and their disappearance can be demonstrated much oftener than a previous change in the fibrillæ. Beyond a certain stage, then, the gravity of a lesion is in proportion to the disappearance of cells rather than according to the kind of changes observable in those which remain. Changes, however, have been described both in the axis-cylinder processes and in dendrons. These are chiefly varicose atrophy which occurs in both kinds of processes, fatty degeneration of the medullary sheath of the axis-cylinder, and an apparent stripping of the ampullæ of the dendrites. Unfortunately, however, much of these may be due to senility, to post-mortem or ante-mortem changes, or to defective preparation. One would expect the earliest changes to occur in the arboriform terminations of the collaterals and in the dendritic terminals, because these are the most plastic and, by all analogy, the most delicate portions of the cell. But if, as seems likely, the structural change, when it does begin in these terminals, is a rapid atrophy, it may easily elude research.

Simultaneously with these lesions of the cell *degenerations of the minute vessels* can be observed, and indications, especially in the subpial vessels, of recent arteritis. Tortuous, distended, unequally bulging, the deep, small vessels have their tunics crowded with proliferated nuclei, and scavenger cells multiply along their course. Miliary aneurysms are abundantly formed, and, under the microscope, the lumen of the small vessels may be seen choked with a waxen-like plug, or the section of the vessel may have dropped out, leaving an empty space. These changes occur both in the white and in the gray matter.

With the shrinking and disappearance of the nervous elements, *sclerosis* increases, and it has a characteristic distribution. In the most superficial layers, immediately beneath the pia in which the deep cell processes are projected, and again in the fifth, large motor cell-layer, the greatest connective tissue infiltration occurs. Under the pia the mass of it is most apparent. There one can often see a dense matting of connective tissue, apparent, when stained, even to the naked eye, and having the appearance, under the microscope, of a dense, wiry web. The spider-cells which have appeared in the subpial zone can be traced down alongside of the inward vessels, and numerous lymphoid elements crowd the perivascular spaces. In the fifth cell-layer, even when the superficial sclerosis is not marked, these products of degeneration are obvious—abundant spider-cells and nuclear proliferation, following the course of diseased vessels and gathering round the decaying cells.

Of perhaps secondary importance we must also note chronic inflammatory changes in the meninges accompanying the endarteritis, amyloid bodies, sometimes very numerous, in the epicerebral space, and, in very chronic cases, great thickening of the meninges.

In the spinal cord the posterior columns and the lateral columns are chiefly affected. Meningitis, increased vascularity, and sclerosis, not of a systematic form, are described by Bevan Lewis, who also selects the intermedio-lateral group and Clarke's vesicular column as the most frequent seat of cell degeneration. The posterior nerve-roots occasionally participate, but probably not by a spread from the peripheral nerves.

To summarise, we must take note chiefly of the wasting and disappearance of nerve-cells, with fatty and pigmentary degenerations, the arteritis and aneurysmal changes in the vessels, and the sclerosis of the subpial and fifth cell-layers.

The pathological changes that may be present in the various viscera are referred to in the article "Alcohol," and also under their respective headings.

Treatment of Alcoholism.—*Social.*—All authorities are agreed that the treatment of alcoholism should be chiefly prophylactic. It therefore becomes the function of physicians to bring about a state of society in which the risk of incurring alcoholic diseases is minimised. That is as properly a part of public health as is the prevention of infectious disease. But apparently this is a subject which only a few can approach in a scientific spirit. Apathy on the one hand and fanaticism on the other characterise much of the literature upon the question

It is perhaps not within the province of medicine to attempt to revolutionise the habits of a whole nation; but it certainly is our function to interfere with conditions which make alcoholism almost a certainty to many. The moderate spirit of scientific physicians would approve that we should in the first instance direct our energies to the development of a popular opinion, and to the passing of laws, which have a distinct medical reference. For example, no one of authority denies the evil effect of impure drinks, and accordingly the faculty might insist that the sellers of alcoholic drink which contains ingredients which are not supposed to be in it, and which are deleterious, should come under the same condemnation as those who publicly defraud or injure the community by adulterations of any other kind of food or drink. Spirits taken from bond, wines, and beers should be certified free from poison before being offered to the public. But to some individuals, notably children and drunkards, any form of alcoholic drink is in the nature of a poison. It therefore becomes the duty of the profession to devise means to prevent an indiscriminate supply of liquor to such persons. It ought to be a crime to give alcohol habitually to children without medical advice, and such a practice should come within the Acts which relate to cruelty and neglect towards children. The only sure preventive of continued indulgence by drunkards is compulsory detention in a home. The faculty has for long insisted that a measure to make that legal is a necessity. But perhaps we may arrive at it better by easy stages. The last Inebriate Act made it legal to detain those who, being habitual drunkards, commit grave crime, or who are four times within a twelvemonth convicted of petty drunken offences. A useful measure would be one which would entitle the bench to convict a person of habitual drunkenness on the evidence of medical and other witnesses by private, or, if preferred, by public trial, and thereupon to bind the patient over under a caution to abstain from drink, and to report himself to the authorities periodically for a term. Failing compliance with such orders the patient might then be liable to compulsory detention; and any one who had supplied liquor knowingly to such an one should be liable to fine or imprisonment. Such forms are suggested, not because they are widely approved, but because it seems proper that a work on general medicine should point out to the profession the kind of measures which it may be their part to busy themselves with. At all events it should be recognised as a principle that our first duty as physicians is if possible to devise measures which are for the prevention and treatment of disease, but which do not interfere with the habits of the multitude who are not, and who are not likely to become, the subjects of alcoholism.

Personal.—General.—The general treatment of an alcoholic patient refers chiefly to the choice of a place where he will reside. If any possibility offers to make it feasible to send him to a hospital or retreat, such a procedure is nearly always best. Although inebriate homes and asylums can at the best only commence the patient's cure, discipline and certain sobriety can be there obtained which are very difficult if he remain at large. But if there are reasons why such a course cannot be followed, much good is often obtained by boarding the patient with some one who will control him. Almost no case can be successfully treated at the patient's own home. On the other hand, it is usually difficult to find a house where one can be sure that treatment will be what one desires. An out-of-door life is in my opinion essential for simple alcoholism.

Where the case is complicated by paretic symptoms a hospital method of treatment is essential; but even then the fresh-air plan should be followed as much as possible. The one necessity is that those in charge of the case should be in a position to enforce the doctor's orders, and that they should conscientiously do so. It is often, for example, necessary to take physical control of a patient, to restrain him perhaps by locking him into a room. As that is illegal, the person in charge must be protected, and it is the duty of the physician to see that every emergency is provided for. Nothing should be left only to the patient's good intention. The trustful policy at least in the early stages of treatment is almost never proper. All likely sources of relapse should be controlled—letters inspected on being opened by the patient, clothes and luggage searched, private interviews with friends or strangers forbidden.

Having disposed of the patient somewhere where treatment can be furthered, there are certain measures which can be carried out with advantage in nearly all cases. These are designed to promote recuperation in the cortical mechanisms and in other organs, and are simply a general plan of hygiene. Metabolism should be increased and excretion pushed by as much open-air exercise of an interesting kind as will just not unduly fatigue, by a somewhat generous diet which is not rich and which is less than usually nitrogenous, by large quantities of liquid, by daily warm baths, and if necesssary by drugs which help the excretory processes. At the same time tonic treatment must be carried out. Hours of food and of sleep must be regular. The heart must be watched and its action assisted if necessary. Frequently anæmia must be combated, and the nervous system must be toned up by the usual nerve tonics. It is too often forgotten that sensory stimuli of all sorts which do not approach severity of shock, and which do not exhaust by being unduly prolonged or too massive, are the natural tonics of the cortex—cold showers, skin friction, and the manifold energies of bright and open surroundings. All such measures can hardly be applied without a special attendant or companion. It is futile to hand an alcoholic over to a man or woman whose time is almost fully occupied in necessary business or domestic details.

Particular.—Special symptoms call for special treatment in alcoholism as in other diseases. Indeed, it is a disease in which it is no disparagement to confess that we are treating symptoms. There is no possibility of directly treating the lesion which is the organic basis of the complaint. At the same time a great deal is done to promote recuperation if we carry out the suggestions just concluded. Space forbids that we should consider each symptom in detail. It is only possible to consider those which usually offer the most serious difficulty.

In breaking the alcoholic habit—and that is a necessary step whatever the complications may be—the question nearly always arises as to whether it should be done suddenly or gradually. A considerable body of opinion, epecially in America, condemns the abrupt method. Perhaps the American constitution is not suited to it. In this country evil consequences are uncommon. We need, therefore, have no hesitation in recommending an abrupt withdrawal of alcohol in every case in which there is no collapse. Only, we must watch the patient very closely for the forty-eight hours immediately following. Even if the pulse should fail, or if a nervous depression be imminent, these can usually be met without resorting to alcohol. Digitalis, strychnin, caffein, and other stimulants are then of great value. But under no circumstances should the patient be told what is being administered. That injunction applies to the whole of the drug treatment of alcoholism. *To treat the volitional defect* which has been occasioned by the alcoholic habit it is convenient to have regard to two aspects of volition—discipline and incentive. In the first instance the whole life of the patient must be methodically ordered. He must keep quite regular hours, and the conventional rules of dress, manner, and general conduct must be strongly insisted on. When the patient has recovered from the immediate effects of alcohol this régime must be particularised. A time-table of occupation should be given to the patient, and he should be made to carry it out however great his disinclination. It is the character of alcoholism that the patient does not do what he has deliberately resolved to do. The further stage refers to the development of incentive. What that may be depends largely upon the individual. But obviously it is not enough simply to develop a disciplined character in the patient. For example, it is the disability of treatment in an asylum or retreat that the later requirement cannot easily be met. We must leave it to the clergyman, or to a relative, or to the inspiration of a public life to teach our patient some large interest which will be at once an impulse and a conscience. A very great value lies in the power which attaches to a personal influence. For nearly every alcoholic some one can be found who will exercise a profound and effective influence over him. That person is often not of the patient's household. In many cases some kind of suggestion is valuable. I have known a case in which the surreptitious introduction of permanganate of potash into the patient's urine and vomit effected a saving fear of internal hæmorrhage. Not a few cases have been successfully treated by graduated injections of apomorphia, which are made to appear to induce a sickness which only follows upon drinking. A great future in my opinion lies before hypnotism in the treatment of alcoholism. It has a therapeutic value which is not sufficiently utilised by the physician who is not a specialist. Some cases are amenable to hypnotic treatment to a remarkable degree, and, after the first experience of it, are really "cured." *When the memory is impaired* it is wise to educate it specially by appropriate exercises, and in particular to teach the patient to observe carefully, and if necessary to take a note of facts until he discovers what things he is most likely to forget, and then to devise an effective reminder. *Sleeplessness* is often a troublesome symptom. Paraldehyde is very useful, but patients are extremely apt to develop a paraldehyde habit. That indeed applies to all sedatives. But it is especially true of drugs which have a pronounced taste or smell. All drugs, therefore, should be given in masked form and unnamed, or by the tube. Experience teaches that each case of sleeplessness must be treated on its merits, and that various drugs may be tried until one is found which suits the patient well. Before resorting to hypnotics, however, a warm drink at bedtime, with a stimulant, should be tried. A little digitalis or strophanthus and strychnin often allows the patient to sleep. Sal volatile should not be given in a recognisable form. Morphia is often particularly useful in allaying the restlessness which can almost amount to pain; but bromide, in large doses, is the most universally useful drug. For the rest, the treatment applies chiefly to *complications.* The gastritis, and other abdominal disorders, nervous, cardiac, and pulmonary complications should be treated *secundum artem*, and with as close conformity to the general principles of the treatment of alcoholism as is possible. Experience proves that a great number of alcoholics recover very quickly from these complications as soon as the alcohol is eliminated.

ETIOLOGY.—It is easy to state simply the problem which presents itself to the student of the causation of alcoholism; but the reply to the questions propounded must always be complex and difficult. There are two aspects of the subject, which we may state in the form of interrogatives: (1) Why do many persons drink to excess? and (2) Why do some of such persons incur the complex of neuroses, to which we give the name Alcoholism? In reply to these queries it is usual to speak of predisposing and of exciting causes, but as a rule the same factor plays both these parts.

It is not enough to say that people drink to excess because they like drinking, for the simple reason that excess obviously does not add progressively to pleasure. Nor is it the case that we can in many instances plead a special "crave" which is characteristic of the drunkard's constitution, though such a factor must be recognised in a few (see "Dipsomania"). We are therefore brought back to the fact that it is a defect of control, an impairment of the voluntary inhibitory functions, which accounts for alcoholic excess. That occurs, as we have seen, as the result of alcoholic dissolution. But it is in many cases a defect which stigmatised the previous constitution and character. For practical utility the practitioner must be on the outlook, especially in the young, for what is well described in the popular term "slackness."

Factors which determine an original or an acquired defect of purpose.

Heredity is of very great consequence, but we must observe that facts are wanting which establish the belief that an alcoholic pedigree is of greater importance than other neuropathic factors (Imbecility, Insanity, Epilepsy, Paralysis, Prostitution, Crime, etc.).

Bodily Distress very frequently induces drinking, especially in middle-class women. We must in particular note the importance of all the evolutional and reproductive incidents (adolescence, pregnancy, etc.), and the abiding lassitude of slow disorders (especially heart affections, neurasthenia, and influenza).

Mental Distress in any form—financial, social, or domestic worry—incites the patient to seek indifference in drinking, and these, and perhaps a commencing insanity, may impair his control.

Factors which aggravate the temptation to drinking.

Constitutional susceptibility to the effects of alcohol is important, as when the patient reacts with unusual delight to stimulants; but this must be distinguished from a specific crave which precedes drinking, and is not satisfied otherwise than by alcohol.

Public Sanction increases the chances of excess, both by the multitude of licensed houses and by the slackness of public opinion.

Domestic Conditions are probably still more important. Squalor necessarily induces want of care, and that and the general irregularity of a drunkard's home are in my opinion as important in educating his children into drunkenness as are their constitutional proclivities.

Factors which determine the pathological alcoholic changes.

Excess of Alcohol, which is a variable quantity for each individual, will induce alcoholism even if the liquor has been pure.

A neuropathic diathesis is of special importance in determining the more aggravated and anomalous effects of alcoholism (Insanity, Epilepsy, etc.). Speaking generally, a patient who reacts unduly to alcohol is likely to incur alcoholism, but many cases have not manifested any notable signs of special neurotic constitution.

Impure Beverages induce all the graver neurotic and visceral disorders in alcoholism. Fusel oil, furfurol and the essence of absinthe are convulsant poisons. The aromatic adulterations are of the first importance in headaches, vertigo, and general depression. The pigments commonly used in wines, and these and the adulterations of beers combine with the impurities mentioned in aggravating the disorders of the stomach, liver, and kidneys. That such impurities are permitted is characteristic of our public indifference, and an unpleasant sign of the perhaps unconscious corruption of those who are responsible for our fiscal policy.

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Aleppo Boil. *See* Furunculus Orientalis.

Alimentary System.—General Index of the main articles dealing with its medical and surgical affections. Consult also outline of "Tropical Diseases."

Abdomen, Clinical Investigation of.
Abdomen, Diagnosis of Abdominal Tumours.
Abdomen, Injuries of.
Abdominal Abscess.
„ Aneurysm.
Appendix Vermiformis.
Appetite.
Ascites.
Breath.
Colic.
Colon.
Colotomy.
Constipation.
Diaphragm.
Diarrhœa.
Diet.
Digestion.
Dyspepsia.
Enteroptosis.
Gall-Bladder and Bile-Ducts.
Hæmatemesis.
Hernia.
Intestine.
Jaundice.
Laparotomy.
Liver.
Mesentery.
Mouth.
Œsophagus.
Omentum.
Pancreas.
Peritoneum.
Rectum and Anus.
Salivary Glands.
Stomach.
Tongue.
Vomiting.

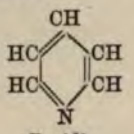
Alkalies and Alkalinity. *See* Blood, Digestion, Urine.

Alkaloids.—Definition.—*Alkaloids* are bodies of considerable molecular complexity, but of definite chemical constitution, formed by the metabolic changes of living protoplasm in the cells of animals, plants and fungi. Their solutions have an alkaline reaction; they combine with acids to form salts; invariably contain the elements carbon, hydrogen, nitrogen, and, with few exceptions, oxygen; and answer as a class to a series of special tests. Like the metallic bases they are electro-positive on being electrolysed. Several of the alkaloids have been produced synthetically.

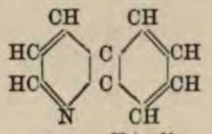
Composition and Character.—Alkaloids as a rule are crystalline or amorphous solids, but some are liquid. They are soluble only with difficulty in water, the resulting solutions possessing a bitter taste; almost all their salts are easily dissolved by water; the alkaloids themselves can be separated from these solutions by the addition of a caustic alkali, or in some cases of an alkaline carbonate. All the members of the alkaloid group form insoluble compounds with the same reagents, such as tetrachloride of platinum, phospho-molybdic acid, tannic acid, and mercuric perchloride along with iodide of potash.

The chemical constitution of the vegetable alkaloids used in medicine has a profound influence upon their pharmacological action. Many of them are pyridin (C_5H_5N) or chinolin (C_9H_7N) derivatives; in fact they are compound ammonias, in which hydrogen atoms have been replaced by various radicals.

To illustrate the modern opinions as to the molecular constitution of alkaloids, the following series of progressive changes in the benzene nucleus (C_6H_6) may be traced. If an atom of nitrogen be substituted for CH in this nucleus, pyridin is obtained, C_5H_5N, or if the nucleus be doubled, and the same substitution made, chinolin, C_9H_7N, is formed.

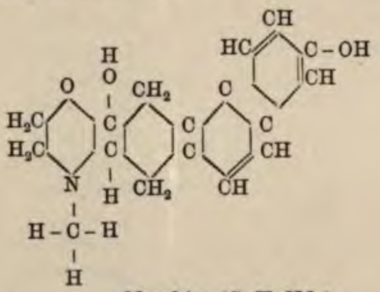


Pyridin.



Chinolin.

If the formula of chinolin be doubled, but without an atom of nitrogen in the additional part, a carbon atom replaced by oxygen, but the carbon reattached to the nitrogen atom in the form of methyl,



Morphina ($C_{17}H_{19}NO_3$)

two hydroxyl radicals in place of two hydrogen atoms, and eight additional atoms of hydrogen, morphina is formed. If a molecule of water be removed, apomorphina results, or, if a methyl radical be joined to the hydroxyl radical shown to the right of the figure, codeina is represented. While thebaina ($C_{19}H_{21}NO_3$), in which a methyl radical is introduced in the place of an atom of hydrogen in morphina, acts antagonistically. Similar substitution of radicals can be effected in the poly-ammonia alkaloids, of which quinina ($C_{20}H_{24}N_2O_2$) and strychnina ($C_{21}H_{22}N_2O_2$) are examples of those obtained from plants; hexatomic owing to the presence of two atoms of nitrogen; and the numerous diamines and triamines formed in the animal body or by the action of fungi.

A striking example of the intimate correlation between chemical constitution and pharmacological action is afforded by many of the alkaloidal bodies. A number of them, although acting in a very different manner when in simple form, exhibit a marked power of paralysing motor nerves when their molecular constitution has been altered by the substitution of the radical, CH_3 (methyl), for an atom of hydrogen. Thus strychnina is a powerful stimulant of the motor ganglia in the anterior cornua of the spinal cord, and is credited with only slight, if any, influence upon motor nerves; methyl-strychnina is a motor nerve and ganglion depressant.

CLASSIFICATION.—

1. *Vegetable.*—A. Elaborated by the living protoplasm of plant cells.
   B. Produced by the metabolic processes of lower vegetable forms.
2. *Animal.*—A. Ptomaines; actually the products of low vegetable forms from dead animal matter.
   B. Leucomaines; formed by metabolic processes during life by animal tissues, as creatin, xanthin, etc.

The alkaloids of vegetable origin belonging to Class 1, A, are those which are made use of in medicine, with a few exceptions, such as, for instance, the obscure alkaloids in cod-liver oil. They may be divided into those which are crystalline and solid—by far the larger group—and those which are liquid, conina, pilocarpina, nicotina, sparteina, etc.

TABLE OF VEGETABLE ALKALOIDS.

A. Solid.

Name.	Formula.
Morphina,	$C_{17}H_{19}NO_3$
Codeina,	$C_{17}H_{18}(CH_3)NO_3H_2O$
Thebaina,	$C_{19}H_{21}NO_3$
Narcotina,	$C_{22}H_{23}NO_7$
Papaverina,	$C_{20}H_{21}NO_4$
Cotarnina,	$C_{12}H_{13}NO_3$
Apomorphina,	$C_{17}H_{17}NO_2$
Atropina,	$C_{17}H_{23}NO_3$
Homatropina,	$C_{16}H_{21}NO_3$
Hyoscyamina,	$C_{17}H_{23}NO_3$
Hyoscina,	$C_{17}H_{21}NO_4H_2O$
Duboisina,	do.
Daturina,	$C_{17}H_{23}NO_3$
Caffeina,	$C_8H_{10}N_4O_2H_2O$
Strychnina,	$C_{21}H_{22}N_2O_2$
Brucina,	$C_{23}H_{26}N_2O_4$
Physostigmina,	$C_{15}H_{21}N_3O_2$
Gelsemina,	—
Conhydrina,	$C_8H_{17}NO$
Cocaina,	$C_{17}H_{21}NO_4$
Jaborina,	$C_{22}H_{32}N_4O_4$
Curarina,	$C_{18}H_{35}N$
Erythrophlœina,	—
Aconitina,	$C_{33}H_{43}NO_{12}$
Aconina,	$C_{26}H_{39}NO_{11}$
Isaconitina,	do.
Veratrina,	$C_{37}H_{53}NO_{11}$
Emetina,	$C_{20}H_{30}NO_5$
Quinina,	$C_{20}H_{24}N_2O_2$
Quinidina,	$C_{20}H_{24}N_2O_2$
Cinchonina,	$C_{20}H_{24}NO_2$
Cinchonidina,	$C_{20}H_{24}N_2O$
Beberina,	$C_{36}H_{42}N_2O_6$
Hydrastina,	$C_{11}H_{11}NO_2$
Delphinina,	—
Scoparina,	$C_{21}H_{22}O_{10}$
Colchicina,	$C_{17}H_{19}NO_3$
Cornutina,	—

B. Liquid.

Name.	Formula.
Conina,	$C_8H_{16}HN$
Methyl-conina,	$C_8H_{14}CH_3N$
Pilocarpina,	$C_{11}H_{16}N_2O_2$
Nicotina,	$C_{10}H_{14}N_2$
Sparteina,	$C_{15}H_{26}N_2$
Lobelina,	—
Lupulina,	—
Pelletierina,	$C_8H_{15}NO$

ACTIONS.—To properly treat of the actions of alkaloidal substances on the body, volumes should have to be filled.

Regarding the subject from the most general point of view, it may be said that the therapeutic employment of alkaloids spells "playing with poisons," and therefore is a matter which properly demands a thorough knowledge of the very many actions exerted by them on a greater or less number of the bodily organs, tissues, and processes, if their administration is to be correctly determined. As a rule, individual alkaloids influence one or other well-defined group of cells or tissue forms in a very prominent degree, the parts remaining often being but slightly affected. One alkaloid may, for instance, affect the cells of the anterior gray matter in the cord, while the cells of the posterior cornua escape untouched.

Table of the Chief Actions of Alkaloids.

1. On the nervous system.

	Stimulated by	Depressed by
A. Motor mechanism.		
Nerve-endings	Morphina (B),[1] nicotina (B), pilocarpina	Conina, atropina, sparteina, cocaina, methyl-alkaloids.
Spinal cord	Strychnina, morphina (B), brucina, thebaina	Eserina, morphina (L), apomorphina, veratrina, emetina, cocaina (large doses).
Brain	Atropina, eserina, cocaina	
B. Sensory mechanism.		
Nerve-endings	Nicotina (B), veratrina (B)	Aconitina, veratrina (L), atropina, morphina, cocaina.
Brain	Atropina, hyoscyamina, caffeina, cocaina, morphina (B)	Morphina (L), hyoscina, cannabinina, lupulina.
C. Secretory mechanism.		
	Nicotina, pilocarpina, eserina, morphina (sweat).	Atropina, morphina, muscarina, strychnina, quinina.
D. On vagus centre.		
Cardiac	Aconitina, veratrina, nicotina, delphinina, atropina (B), hyoscyamina (B)	The same in large doses.
Respiratory	Strychnina, apomorphina	Eserina, codeina, conina, caffeina, quinina, emetina.
E. On accelerating cardiac centre.		
	Caffeina, delphinina.	
F. On vomiting centre.		
	Apomorphina, emetina	Morphina.
G. On vaso-motor nerves.		
	Atropina (B), strychnina, veratrina (B)	Atropina (L), lobelina, nicotina, veratrina (L), emetina, aconitina.
H. On splanchnic nerves.		
	Atropina (moderate doses)	Atropina (small doses).

2. On muscles. Increase power of work, caffeina; diminish this power, emetina, cocaina, quinina, apomorphina.

3. On the blood. Affecting the white corpuscles, veratrina and quinina; the red corpuscles, quinina and allied alkaloids.

4. On the eye. Atropina and its allies, conina, gelsemina, muscarina, aconitina paralyse endings of third nerve; cocaina stimulates the endings of the sympathetic; these dilate the pupil.

   Pilocarpina and nicotina stimulate the third nerve endings, and eserina stimulates the muscle, contracting the pupil.

5. On the organs of generation. Strychnina, cannabinina are aphrodisiacs; most of the opium alkaloids, atropina and its allies, and cocaina are anaphrodisiacs.

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[1] (B) = Briefly; (L) = Later.

Motor nerve endings are stimulated by pilocarpina, briefly by morphina, and slightly by strychnina; the motor spinal centres by strychnina, brucina, and thebaina; and the cerebral motor cells by atropina and physostigmina. The motor nerve endings are depressed by curarina, conina, atropina, nicotina, hyoscyamina, sparteina, cocaina, and a number of methyl compounds of the alkaloids of the preceding group; the spinal motor centres by physostigmina, morphina (later), apomorphina, veratrina, emetina, lobelina (*cf.* table).

Sensory nerve terminations are stimulated by veratrina at first, the sensory and mental centres in the brain by atropina, hyoscyamina, cocaina, caffeina, and morphina (briefly); the sensory nerve endings depressed by aconitina, veratrina (later), atropina, morphina, and cocaina; the cerebral centres by morphina (later), hyoscina, cannabinina, lupulina.

The alkaloids which act upon the vaso-motor and secretory systems influence the nerve-fibres themselves or their terminations, the local ganglia, the various secretory centres in the cord, and the vaso-constrictor, or the vaso-dilator, medullary centres.

Thus pilocarpina, muscarina, and nicotina are sialagogues by local action, atropina and hyoscyamina anti-sialagogues; sparteina contracts the renal afferent vessels locally, caffeina acts either on the renal cells or on the nerves ending in them. To illustrate the actions of alkaloids upon the vaso-motor centres, the dilatation of blood-vessels by atropina, conina, lobelina, their contraction by cornutina and strychnina may be adduced; while nicotina acts as a diaphoretic by influencing the sweat centres.

Quinina and veratrina lessen the motility of leucocytes; quinina acts also upon the red blood corpuscles, rendering oxyhæmoglobin more stable, and thus diminishes metabolism.

The pharmacological actions of many of the alkaloids, it is worthy of note, are mutually antagonistic; this may be true of actions affecting the whole organism, of one organ, tissue, or one group of cells.

The Law of Dissolution.—The progressive effects of many of the vegetable alkaloids after continued administration for some period of time, or if the dose given be increased, upon the various portions of the nervous system, appear in many cases to follow what has been termed a "law of dissolution"; *i.e.* nervous faculties and powers acquired by man more recently are influenced before those faculties less specialised, and therefore of earlier acquirement. Some drugs act only upon the more recently acquired faculties at first, or affect them more profoundly, then in succession proceed to act more and more upon the less and less specialised faculties and functions until the nervous mechanisms common and essential to all animal bodies are reached. Thus morphina first influences the mental central areas—thought, memory, ideation, are exalted, then depressed; in turn the lower and more reflex centres are affected.

Other alkaloids exhibit in their actions the converse, first altering the conditions of the earliest, then progressively the more recently acquired, nervous organisations. Thus strychnina first stimulates the motor centres in the spinal cord, then, if in large enough dose, stimulates the medullary centres, although it never appears to cause any mental disturbance or affect the motor nerves themselves however large the dose. Atropina, on the other hand, first depresses sensory and vaso-motor nerves, stimulates for a short time and then depresses the great medullary centres, later on acting as a stimulant to the cerebrum.

Another instance of this law of dissolution may be applied to alkaloidal actions upon the animal economy. Many alkaloids which act in man and the higher animals chiefly upon the brain will be found to affect the spinal cord in a more pronounced manner in the case of animals low down in the scale. In these animals but few faculties or powers have been acquired apart from those essential for the mere act of living, so that the centres which opium affects in man so strongly as to obscure the special action of thebaina on the cord, are not so markedly acted upon in the frog as are the motor ganglia in the spinal cord, the action of morphina here approaching that of thebaina in higher forms.

The alkaloids, in fact, appear to be endowed with a species of selective power, acting upon one form of cell or tissue, and not upon another form, even if it be closely allied to the first both in position and function, in terms of a law which is at present inexplicable. Morphina stimulates the secretory nerves governing the sweat-glands, but depresses the secretory impulses transmitted by the same class of nerves to all other secretory glands. Atropina depresses the entire sensory and motor nervous apparatus below the cerebrum, but stimulates the nerve-centres there.

When given in minute doses many alkaloids act conversely to their actions when given in ordinary doses; as an instance, the emetic action of ipecacuanha given in large enough quantity, and the anti-emetic action of the same when administered in a proportionately small dose repeated frequently, may be noted.

As a class the alkaloids which are used therapeutically are prone to produce a marked tolerance in the persons of those who take any of them regularly. Their actions, therefore, soon become less pronounced, and larger amounts have to be given. In some diseases, again, large doses of alkaloids can be administered with impunity, as morphina in peritonitis, quinina in malaria, etc. It almost appears that the alkaloids which are used in medicine—the products of protoplasmic activity—inherit much of the waywardness of their parental protoplasm, and are influenced by it both as regards the character and the degree of their actions upon the protoplasm of cells when introduced into a living animal body.

The alkaloids belonging to other classes are chiefly of interest from a bacteriological aspect, or with regard to digestive processes and intestinal decomposition. A consideration of their properties would lead us too far afield here. In their chemical reactions they correspond closely to ordinary vegetable alkaloids, in their actions on the body they present great contrasts; some are powerful toxic poisons, others are almost inert.

The fate of alkaloidal substances in the body is an interesting but obscure subject. Some are excreted unchanged, as morphina, conina, and atropina; others are altered during their transit, so strychnina partly appears in the urine as strychnic acid, cocaina as ecgonina (partly at least); while the composition of animal alkaloids absorbed from the bowel is altered in healthy persons in the liver, and excreted in a non-poisonous derivative form.

Alkaptonuria. *See* Urine.

Allantiasis. *See* Food Poisoning.

Alopecia.

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Alopecia is the term applied to baldness arising from any cause. The chief varieties of baldness are usually classified under three headings, congenital, senile, and premature. The name alopecia areata is used to signify a special form of baldness, and will be described later.

Congenital Alopecia.—This is a rare condition, and when present is often associated with other deformities of the skin. It may be universal or in patches. In connection with the latter form it is important to recollect that the distribution of the hair varies in different human races. Some of the varieties of patchy congenital baldness may be related to inherited developmental characteristics. It is not uncommon to find a scanty development of hair as a family peculiarity.

Senile Alopecia.—The shedding of hair which occurs as the consequence of age is caused by the failing nutrition, from which all organs suffer, and which produces characteristic changes in the skin. The alterations in the elastic tissue which are peculiar to the senile skin, and the gradual atrophy of the other skin structures, show the want of full nutrition and the failure of the function of the tissues, and one of the consequences is that the hair is shed. This change shows itself, as a rule, first in the scalp, the skin of the vertex being first affected. The area of baldness increases till only a fringe of hair may be left at the margins of the scalp. Simultaneously with the loss of hair on the vertex, the baldness frequently advances on each side from the frontal region, leaving at first the central crest of hair unaffected. Gradually the hair of the central region also falls. More rarely the shedding of the hair is general from the commencement. The loss of hair may be noted over the whole body, due to the same causes.

Premature Alopecia.—Baldness especially of the scalp occurs also in young persons, and may progress to such a degree as to cause almost total baldness before the age of thirty; not infrequently it may be seen to commence at about the age of twenty. This condition is much more common in men than in women. In such cases the loss of hair occurs very markedly at each lateral aspect of the frontal region of the head, and spreads backwards to meet a central area of baldness commencing at the vertex. It is in the case of this variety of alopecia that the central longitudinal crest of hair is most frequently left. It is often noticed that a finer and less pigmented growth of hair takes the place of that already shed, which in its turn also disappears, till complete baldness results. After advancing for a certain period this form of the disease often stops, while the hair on the lateral aspects of the scalp remains quite thick and of normal colour.

There can be little doubt that many of the cases of premature baldness are the result of *seborrhœa* of more or less severity. Very often it is a sharp attack of seborrhœa capitis at about the age of twenty or twenty-five which is the starting-point of the trouble, and usually other symptoms of seborrhœa, such as pityriasis, greasy skin, over-sweating, or even dermatitis seborrhoïca, are also present. In such cases the loss of hair during the first few weeks is excessive, but as the seborrhœic process diminishes, the loss of hair becomes less and may cease altogether.

Other varieties of Alopecia.—There are certain cases in which *no symptoms of* distinct *seborrhœa* can be distinguished, though the actual loss of hair may be even greater than in the seborrhœic variety. Little is known of the etiology of this non-seborrhœic alopecia, except that it frequently seems to occur in families. In families who have the tendency to premature alopecia of this form the females may be as severely affected as the males. The prospect of recovery of the hair varies considerably in the group of cases of premature alopecia referred to. If the loss of hair is associated with seborrhœa, the prospect of the hair growing, at any rate to some extent, is good if the latter disease can be rapidly checked, but in cases of seborrhœa of long duration, when the hair papillæ have become atrophied, and especially in the non-seborrhœic variety, the prospect of regaining the hair is very slight.

Alopecia may arise also as the result of many forms of disease, and is temporary or permanent, rapid in its onset or gradual, according to the nature of the predisposing malady. It arises very commonly as the result of severe illness, especially of the *infective fevers*, such as enteric. It is very usual as the result of erysipelas, especially if the inflammatory process has affected the hairy regions such as the scalp. In such cases the recovery of hair is often very rapid. Occasionally, however, a condition of slight chronic inflammation of the skin, accompanied by pityriasis, persists, which delays the recovery of the hair, and which may itself become a cause of baldness.

Gradual alopecia is common in many forms of *chronic and debilitating disease*, such as in pulmonary tuberculosis, diabetes mellitus, myxœdema, etc.

The influence of the *nervous system* on the nutrition of the hair papillæ is occasionally shown in cases of sudden, universal or local alopecia following mental anxiety or sudden shocks.

Localised inflammations of the skin frequently result in loss of hair with temporary or permanent baldness. Thus, it occurs especially on the scalp in cases of impetigo, eczema, psoriasis, trichophytosis, etc. In such cases the permanence of the baldness depends, as a rule, upon the amount of destruction of the hair follicles which is produced, and this effect is most common when suppurative folliculitis occurs, *e.g.* as the result of trichophytic infection of the scalp. Such diseases as lupus erythematosus, folliculitis decalvans (necrotica), and affections producing ulceration of the surface, such as tuberculosis of the skin, syphilis, and leprosy, naturally cause baldness, as the result of the destruction and cicatrisation of the skin which they produce.

The various forms of *sclerodermia* produce baldness of a special character, which may be distinguished by the characteristic alterations in the appearance and texture of the skin which are present. In the case of the scalp the form of sclerodermia usually seen is the localised sclerodermia (morphœa), which may be observed to occur in the areas of nervous distribution in a very characteristic manner.

Syphilis is a frequent cause of baldness. It may do this on account of its debilitating effects as a general constitutional disease, or it may produce the effect by its local manifestations. The baldness so produced may be permanent, as when atrophy or cicatrices of the skin result. A peculiar variety of localised baldness is not infrequently seen in the course of syphilis, without visible signs of inflammation of the skin; and this condition may closely simulate the baldness of alopecia areata. It may usually be distinguished, however, by the absence of definite limitation of the patches, by the tendency to diffuse alopecia which usually coexists, by the retention of the normal texture of the skin owing to the persistence of the hair follicles, by considering the history of the case, and frequently on account of the rapidity with which the hair recovers on anti-syphilitic treatment.

General alopecia is a rare affection, the etiology of which is practically unknown. Usually between the ages of eighteen and twenty-five there is a general loss of hair over the whole surface, so that in the course of a few months the body is deprived of hair even to the destruction of the fine lanugo-hairs. In perhaps the majority of the cases a few weak, slightly pigmented, hairs may remain on certain parts of the body, so that while the scalp, the face, and the greater part of the trunk may be quite bare, occasional hairs may be seen in the axillæ and on the pubic region. Occasionally also lanugo may remain here and there on the trunk. It is usual in such cases to observe a certain degree of atrophy of the skin and the hair follicles, and on regions such as the scalp the latter may be almost completely obliterated. Frequently also the general pigmentation of the skin is diminished. The prognosis in such cases is bad, the hair being recovered even in favourable cases only in patches.

A patchy variety of alopecia is occasionally, although very rarely, caused by *injuries*. The injuries (apart from actual destruction of the skin by burns, etc.) which have been noted as the precursors of this variety of baldness are such as have involved the nerves. The consequent baldness appears in the distribution of the nerves presumed to be affected; thus, a blow from a cricket ball on the left ear was followed by severe neuritis and a bald patch in the left parietal region. Injuries in the neck and in the neighbourhood of the carotid sheath have been succeeded by baldness in areas of the scalp corresponding to injured nerves. So also sufferers from neuralgia sometimes develop patches of alopecia in the distribution of the affected nerves. Such cases as these support a certain amount of experimental evidence advanced by Joseph and Mibelli, who attempted to produce baldness by excising portions of nerves.

Under the name of *alopecia circumscripta* a peculiar form of baldness has been mentioned, in which the loss of hair occurs in small patches, usually depressed below the surface and with a certain amount of loss of sensation. In such cases the hairs become loose and can be detached readily; trophic alterations of the nails, such as change in colour, thickening, deformity, and separation from their beds, are described, and there is no appearance of the gradual atrophy of the hair so characteristic of true alopecia areata.

Alopecia areata in its true form is usually readily distinguishable from the above-mentioned varieties of baldness. It is this disease which gives rise to by far the larger number of cases of patchy baldness. According to Crocker's statistics it forms about 2 per cent of all forms of skin disease in England, 1·5 per cent in Scotland, and 3 per cent in France.

Symptoms.—The disease is usually noted when it has already produced a small round or oval patch of baldness on the scalp. It is on account of the absence of premonitory symptoms that the actual commencement of the first patch is usually not observed. The patch, as it usually comes under observation, consists of a surface of remarkably smooth bald skin usually on the scalp. Its edges are well defined, and while the central portion is perfectly smooth, devoid of hair, and shows the mouths of the hair follicles often somewhat diminished in size, the margins of the patch show hairs in actual process of atrophy. These short hairs have a peculiar shape very characteristic of this disease. The shape is that of a point of exclamation or a minute Indian club. This peculiar appearance is due to the fact that the hair is no longer being produced healthily from the papilla, so that the youngest portions nearest the scalp are thinner and often less pigmented than the older portion at a slight distance from the scalp. In process of time the papilla refuses to produce a hair which can be projected from the follicle, and in this way the skin becomes bald. The patches of baldness become usually multiple, sometimes being quite numerous, but in nearly all cases the disease commences as a single patch. They may increase to one or two inches in diameter, and then are likely to coalesce with neighbouring patches, and in this way areas of baldness with serpiginous outline are produced. In addition to the rounded areas usually formed, the disease occasionally has a linear distribution; a very characteristic form of it is that commencing at the periphery of the hairy scalp and encroaching upwards all round, at length producing a band of baldness round the head. The peculiarity of the physiognomy so produced by the loss of hair—a bare band above the ears and round the occipital and frontal regions—is very readily recognised.

Alopecia areata need not affect the scalp only. It is frequently found on the face, producing characteristic baldness of the beard or moustache, and may occur, though less commonly, elsewhere on the body.

The progress of the disease is very variable; in some cases, perhaps the majority, a few patches only are produced. In other cases the disease may become so extensive as to produce almost complete baldness. In certain cases the baldness may last for a few weeks only; in other cases it may persist for months or even years, and the hair still be reproduced; in still other cases the baldness produced is permanent. It is stated that if the baldness lasts for over two years the likelihood of recovery is but slight. At the height of the disease the appearance of the bald patch is very characteristic. We observe the central perfectly smooth surface stippled over with the mouths of the hair follicles, which become smaller the longer the disease persists. This central region appears in chronic cases to be slightly depressed below the surrounding surface, and seems less pigmented than normal, and in some cases appears to show slightly diminished sensibility. On the periphery, the short point of exclamation, hairs can be seen. As the patch enters on the stage of recovery the point of exclamation marks are no longer visible; over the affected area a growth of fine unpigmented lanugo is observed. This fine hair gradually increases in strength and in pigmentation till, in favourable cases, the hair becomes perfectly restored. Occasionally the first crop or even succeeding crops of lanugo may be shed, the perfect growth becoming established only after several attempts. In certain cases of the disease, the patches never become perfectly bald, but produce throughout the duration of the attack an irregular growth of short, weak, slightly pigmented hair. This condition may last for some time, and may either end in diminished production of hair, and the occurrence of complete baldness, or the case may progress satisfactorily, the production of hair becoming gradually more normal in its character. The cases in which the lanugo persists are, as a rule, favourable, but occasionally the growth of the lanugo may persist for years, repeatedly forming and falling, and after all terminate in complete baldness.

HISTOLOGY.—In parts where the disease is well established there is usually found on microscopic examination of the skin slight infiltration of round cells in the neighbourhood of the hair papilla and follicle. Within the follicle itself it is possible to observe more than one fine hair lying bent or curved upon itself, being unable to emerge from the follicle. In such cases the papilla is usually small and shrunken in size. In cases ending in permanent baldness the round-cell infiltration passes on to the formation of new fibrous tissue, so that the papillæ and skin show cicatricial changes which account both for the destruction of the papillæ and hair and the slightly atrophic character of the skin. Recently the histology and etiology of alopecia areata have been reinvestigated by Dr. R. Sabouraud in Paris.

This observer describes the bacterium which had been previously observed by Unna, Hodara, and Engmann in the interior of comedones, and by many observers in cases of seborrhœa oleosa as the specific organism of alopecia areata. This organism is a minute bacillus, and is found to grow in enormous numbers in the mouths of the hair follicles, especially at the margins of the patches, in alopecia areata. As a result of this growth there is formed a cocoon-like mass of desquamated epithelial cells, sebaceous material, and bacilli occupying an ampulliform dilatation of the upper part of the hair follicle described by Sabouraud as the "utricle peladique." In this ampulla, and in uncomplicated cases of the disease, the micro-bacillus exists in pure culture. As a result of these alterations and obstruction at the mouth of the follicle, and possibly owing to the toxins produced by the micro-bacilli themselves, the hair papilla undergoes atrophy, the hair falls out, the papilla continues to attempt to produce new hairs, which are weaker and still weaker and finally fail to emerge from the follicle. In consequence the lower part of the hair follicle may contain several minute recurved hairs, the results of repeated growths from the papilla. Sabouraud has succeeded in cultivating this organism, and describes its characters. He arrives at the conclusion that it occurs both in seborrhœa oleosa and in alopecia areata and is presumably the cause of both diseases. The external evidence by inoculation has not yet been offered, but nevertheless Sabouraud ventures the conclusion that "seborrhœa oleosa and alopecia areata are essentially identical processes." The patch of alopecia is nothing else than an attack of acute circinate seborrhœa, and conversely bald persons would not have become bald but for a diffuse attack of chronic alopecia areata. In a later communication the same author declares that the specific micro-bacillus of seborrhœa oleosa, when it gains access to one of the pilo-sebaceous follicles, produces within it four constant results:—

(*a*) Hypersecretion of sebum.
(*b*) Hypertrophy of the sebaceous gland.
(*c*) Progressive atrophy of the papilla.
(*d*) Death of the hair.

It will be observed, however, that the evidence in support of the specific action of the micro-bacillus is by no means complete, and that Sabouraud brings together under one category, cases of seborrhœa oleosa with true seborrhœic baldness and alopecia areata, conditions which most observers still hold to be distinct. The final results of the process which give rise to atrophy of the papilla and the production of new fibrous tissue in its neighbourhood are confirmed by Sabouraud's researches.

It must be mentioned also that parasites belonging to the group of the hyphomycetes have been described in the hair follicles of cases of alopecia areata. Recent researches into the mycology of ringworm have shown that certain varieties of the trichophyta produce areas of circinate baldness closely resembling the lesions of alopecia areata; careful examination, however, of these patches of so-called "bald ringworm" shows the presence of trichophyta in the usual positions, and there can be little doubt that the cases of alopecia areata in which hyphomycetes have been seen are examples of this exceptional form of ringworm.

The clinical evidence in favour of true alopecia areata being a disease due to the action of a parasite is considerable, and is based on its method of growth, and from certain observations which have been made on the occurrence of the disease, or a condition closely simulating the disease, simultaneously in members of the same family, and in a few small epidemics. Many of these cases no doubt have been examples of the form of "bald ringworm" already alluded to; still, a certain amount of evidence and the opinion of many careful observers are in favour of the parasitic origin of the disease. There are many difficulties in the way of accepting Sabouraud's statement that the micro-bacillus described by him is the specific cause, apart from the absence of experimental evidence, and his hypothesis is not accepted by clinicians nor by most histologists. At the present time, therefore, we are not in a position to identify the parasite which is probably concerned in the production of true alopecia areata.

Prognosis.—The prognosis in cases of alopecia areata varies greatly. In young persons, and when the disease is in patches, recovery nearly always takes place; on the other hand, if the disease has affected large surfaces and has lasted for two years, perhaps with repeated and gradually lessening attempts at recovery, the prognosis is bad. Such cases occur often in elderly people, and the affected scalp may be observed to be slightly and superficially wrinkled, and atrophied, while the hair follicles are scarcely visible.

TREATMENT.—Success in the treatment of alopecia depends greatly on the character of the case. In many of the varieties of the disease mentioned above, treatment is of little avail, as in cases of senile alopecia, alopecia circumscripta, and general or diffuse alopecia. On the other hand, in alopecia following acute fevers, and in some varieties of syphilitic alopecia, the natural progress is towards recovery, a tendency which may be greatly hastened by appropriate treatment. In all cases of doubtful causation every care must be taken to improve the general nutrition of the patient, and underlying conditions, such as syphilis or myxœdema, will obviously require appropriate treatment.

The form of alopecia most amenable to treatment is that produced by seborrhœa, and as this condition complicates many of the varieties of alopecia, it always claims attention. In all cases in which seborrhœa is present it should be kept in check.

Directions should be given to wash the scalp or affected skin frequently. A soap lotion, *e.g.*

℞. Saponis mollis	℥viij.
Spirit. myrciæ acris	ʒij.
Alcohol (90 %)	ad ℥xij.

M.

may be prescribed. From one to three ounces is mixed with half-a-pint of warm water and used to wash the scalp; the soap should be washed completely out of the hair, and both hair and scalp dried thoroughly. The hair and skin should not be left in this state, as the hair becomes brittle and the skin too dry or even irritated, but some form of oil should be used and applied to the skin with friction. With the latter application the drugs found useful in the treatment of alopecia can be readily incorporated. Of these remedies those found to be of most service are sulphur, resorcin, and the salts of mercury. A pomade for the scalp may be used, such as—

℞. Resorcin	grs. 5 to 20	1%— 4%
Sulphuris precipitat.	grs. 10 to 30	2%— 6%
Adip. lanæ hydros.	ʒj. to ʒij.	12%—24%
Paraffin albi moll.	ad ℥j. to 100 pts.	

M.

A small amount of almond oil should be added if the wool fat makes the preparation too tenacious, and aromatic oils may be added if required. Care must be taken in carrying out this treatment not to produce an inflammatory reaction in the scalp or skin; the cleansing and antiseptic effects only of the treatment are desired. The amount of the potash soap, the strength of the ingredients in the salve, and the frequency of the application can be varied so as to regulate the result. Frequently the use of an ointment such as the above is objected to, especially by persons with thick or long hair, on account of the discomfort produced by its greasiness. As a useful alternative the drugs may be used in the form of a lotion, *e.g.*

℞.	Olei ricini *vel* olei amygdalæ	℥ss.	10%
	Resorcin	ʒiss.	1%— 2%
	Ol. rosæ, ol. neroli, etc.	gutt. v.	
	Alcohol (90 %)	ad ℥vj. to 100 pts.	

M.

The amount of oil may be increased if necessary, and other drugs may be combined with proper pharmaceutical precautions. In place of solutions of soap, preparations containing borax, quillaia saponaria, saponin, etc., are frequently of service.

In addition to the anti-seborrhœic treatment outlined above, applications containing counter-irritants are recommended and widely employed. By their use it is desired, by producing a certain degree of erythema of the affected scalp, to increase the nutrition of the hair papillæ. This method of treatment must be used carefully, so as not to produce an inflammatory condition of the cutis, which will obviously do more harm than good. The remedy most used to accomplish this is cantharides, which is the active ingredient of many of the "stimulant" scalp lotions. Such a prescription as this may be used:—

℞.	Tinct. nucis vomicæ	℥ss.
	Tinct. cantharidis	ʒiiss.
	Glycerini	ʒiiss.
	Acidi acetic.	℥ss.
	Aq. rosæ	ad ℥vj.

M. Ft. lotio.

To be applied gently to the scalp night and morning. (After Tilbury Fox's prescription.)

Many other remedies are used for producing the same effect, such as solutions of ammonia, solutions of acetic acid, volatile oils and similar preparations, such as the oil of cade, ol. pini sylvestris, ol. betulæ pyrolignei, oil of rosemary, capsicum, etc.

Other remedies in use for similar purposes have useful antiseptic properties, such as phenol, resorcin, chloral, quinine, and perchloride of mercury. Jaborandi and its alkaloid pilocarpine have been much used on account of their physiological effects on the skin, but with very variable and doubtful results.

The treatment of alopecia areata is carried out on similar principles to those already noted. It is very important to use anti-seborrhœic treatment vigorously in the majority of the cases of the disease, and frequently the best results will be obtained when using sulphur or resorcin, or a combination of these. The "stimulant" treatment has been especially vigorously used in this affection, even to the extent of repeated blistering of the scalp, but there is little doubt that more harm than good is likely to be done by such methods. The counter-irritation should not proceed beyond the rubefacient stage. In hospital practice the use of the lin. terebinthinæ aceticum, to which may be added acet. cantharidis, from ʒj. to ʒij. to the

℥j., proves very satisfactory; similar preparations made with greater pharmaceutical elaboration may also be employed. The use of jaborandi is stated to have produced good results in certain cases of alopecia areata. The tincture is used internally, commencing with small doses, and increasing to the limit of tolerance of the patient; but better effects seem to be attained by the hypodermic injection of the salts of pilocarpine (hydrochloride, nitrate, salicylate) into or near the affected areas. The treatment is commenced by using $\frac{1}{30}$th of a grain in each injection, and the amount of the alkaloid is gradually increased till the full physiological action of the drug is produced.

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Alveolar Abscess. *See* Jaws, Diseases of.

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Amblyopia literally means defective, weak, or blunted vision (ἀμβλύς = blunt, feeble, and ὤψ). While the term amaurosis (literally blindness; ἀμαυρός = obscure, blind) has now nearly become obsolete, amblyopia is still in common use. The former was for long applied to all cases of blindness, to account for which there was no visible change in the eye itself. Since the introduction of the ophthalmoscope, however, the various causes of amaurosis are mostly capable of being diagnosed. Consequently it is no longer the blindness, which is only a symptom, but the various diseased conditions of the retina, optic nerve, etc., which give rise to it, which form the basis of modern nomenclature.

For centuries, too, amblyopia has been the name used to designate defective vision, for which there did not exist any recognisable cause in any part of the eye. And although it must be confessed that the term is not infrequently somewhat loosely employed, this must still be regarded as its proper application. Just, however, as the ophthalmoscope has so enlarged the capabilities of diagnosis as to render the term amaurosis only very rarely applicable, so it has very greatly curtailed the number of those conditions of defective sight which may now correctly be classified as amblyopia. Yet there still remains a considerable group of cases. In this group, too, may be included cases in which more or less evident ophthalmoscopic changes are to be met with, which changes, nevertheless, are of themselves insufficient to account for the symptoms observed, or first make their appearance later.

No doubt a great many causes of amblyopia exist. Not infrequently it is by no means easy to feel sure of the cause. In much that has been written on the subject it is difficult to avoid the suspicion that not a few writers have ascribed many cases to causes which a more critical observer would hesitate to admit. Of such may be mentioned more particularly menstrual and uterine disorders, pregnancy, lactation, etc. Without doubt, the frequency with which some change in the reproductive organs has been associated causally, not only with amblyopia, but with every conceivable form of eye disease, has been very greatly exaggerated. Such changes are of course so frequent that their coincidence, in some form or other, with amblyopia in any particular case, would be at least as likely as not.

We may classify the known and more probable causes of defective vision which, in the sense of the definition given above, might properly be diagnosed as amblyopia as follows:—

A. Toxic amblyopia.
B. Functional and reflex amblyopia.
C. Amblyopia due to retro-ocular (orbital) changes in the optic nerve.
D. Amblyopia due to cerebral changes.
E. Amblyopia due to defective development.

A. Toxic Amblyopia

The poison which gives rise to amblyopia may be introduced from without, or may be elaborated within the body itself. The most important cases belonging to the first category are tobacco amblyopia and lead amblyopia. But there are other less frequent causes of toxic amblyopia which may be referred to the same group. Of these may be mentioned bisulphide of carbon, quinine, salicylate of soda, morphia, santonin, and iodoform. The cases belonging to the second category present, on the whole, more transitory and less characteristic symptoms. The commoner forms of self-poisoning in this respect are uræmia and the digestive disorders which give rise to megrim. Similar symptoms have occasionally been met with after diphtheria and other specific fevers. With regard to diabetes, which has been looked upon as a direct cause of central amblyopia, it is very doubtful whether there is here a self-poisoning in the true sense. The cases themselves are, however, important, and are discussed below.

Tobacco Amblyopia.—This form of amblyopia is of very frequent occurrence. It is met with mainly amongst the working classes. It affects almost exclusively men. The amount of tobacco consumed either by smoking or chewing varies considerably. Most commonly the individuals affected do not smoke less than 3 oz. a week. Although smoking may have been indulged in constantly for many years, it is comparatively seldom that the characteristic symptoms make their appearance before the age of forty. It is not probable that there is any greater inherent predisposition to tobacco amblyopia in men than in women. The relative infrequency of the affection in women seems to depend entirely upon the habit of smoking being so much less common. Indeed the average amount of tobacco consumed is less amongst women who are affected by the amblyopia than it is amongst men thus affected.

Other things being equal, tobacco smoked in a pipe is more liable to lead to amblyopia than when smoked in the form of a cigar. In this country it is extremely rare to meet with tobacco amblyopia in a cigar-smoker. No doubt this is partly due to pipe-smoking being so much more general. Yet the writer has not met with one case in 500 in which cigars only have been smoked.

Predisposing causes.—In addition to age and individual susceptibility, we may recognise certain more or less evident predisposing causes, or perhaps what might more correctly be spoken of as conditions, which lead to diminished resistance to the toxic effects of the tobacco. Sleeplessness, prolonged anxiety, alcoholism, malnutrition of any kind, the state of convalescence after serious illness, are, for instance, all factors which undoubtedly play an important part in this respect. There may have been no increase in the amount of tobacco daily consumed, yet the coincidence of such general conditions with more or less excessive smoking tends in men beyond middle life to admit of the cumulative poisoning which would otherwise not occur in most cases. The habit, too, of smoking on an empty stomach is perhaps the most frequent predisposing cause, in the absence of any malnutrition. It is a very common thing to find working-men smoking the first thing in the morning before breakfast, and therefore before having had any counter-stimulus in the way of food. From the very great frequency with which this habit is met with in those who have toxic amblyopia, it may indeed be regarded as one of the most important indirect causes. It is probably also the most important cause of the relative frequency of tobacco blindness amongst working-men. Other causes, however, of this may be the stronger kind of tobacco smoked, and the greater frequency of chewing.

Symptoms.—Tobacco amblyopia always assumes the form which has been called "central amblyopia." This characteristic of certain groups of cases in which only, or mainly, and to a relatively greater extent, the functions of the central portion of the field of vision become impaired, has long been recognised. Von Graefe, although he did not discover that tobacco poisoning was the cause of a large proportion of such cases, yet very clearly pointed out their importance from a prognostic point of view. Central defects, existing along with a free field peripherally, only rarely proceed to progressive blindness. The opposite is the case when an increasing blindness is found to be associated with peripheral restrictions of the field of vision.

In tobacco amblyopia, then, we find the defect of function limited to the central portion of the field. This occurs, too, always in both eyes. The central scotoma or blind area is never absolute, *i.e.* although the vision corresponding to the particular area is more or less reduced, it is never altogether lost. In this respect tobacco amblyopia, even of long standing, differs from a rather rare affection of the optic nerve, which is also bilateral, but which always occurs considerably before the age of forty, and to which the names "scotomatous atrophy" and "hereditary neuritis" have been given.

The scotoma of tobacco amblyopia is *negative*, *i.e.* it is not projected in space as a dark spot, but merely exists as a circumscribed area of relative blindness of the existence of which the patient is only conscious by finding that objects are imperfectly seen. Sometimes a misty sensation is complained of. This is never of the same decided character as that associated with glaucoma or choroiditis. The mistiness, when it is complained of at all, is most noticeable when facing a strong light. Patients suffering in this way often declare that they see better in the dark. Their vision is, however, only relatively, not absolutely, better when the surrounding illumination is diminished in intensity. A tobacco blind individual, for instance, whose visual acuity is reduced to one-tenth in ordinary illumination, will never present a higher degree of acuity in semi-darkness. On the other hand, as compared with an individual possessed of normal visual acuity, there will be found to be a very much smaller difference when the acuity of both is tested in a light sufficiently bad to interfere with the sharpness of normal sight, than when tested in good daylight.

The scotoma occupies an area of the field which is, correctly speaking, more paracentral than central. Its shape is oval with the long axis horizontal. It stretches from slightly to the inner side of the point of fixation to the blind-spot, and therefore corresponds to the area of the retina lying between the outer portion of the macula and the optic nerve. There is usually a very distinct line of demarcation between the scotoma and the surrounding field. In respect to shape, size, and intensity, there is a remarkable degree of similarity in the scotoma of both eyes. The amount of blindness is, however, not equal over the whole area, but is most complete at the centre at a point which corresponds to that portion of the retina which lies about midway between the fovea and the optic nerve.

The light-sense, colour-sense, and form-sense are all diminished in acuity.

Often before the visual acuity is reduced, as tested in the ordinary way with black types on a white background, the patient complains of difficulty in recognising faces. He is able to read, but finds that external objects present in many cases a smudged, more or less washed-out, appearance. This is due to increasing difficulty in recognising differences in light intensity. Contrast in luminosity has to be much greater than for the normal eye before it can be properly appreciated. The various features of a face are not, under ordinary conditions, very greatly differentiated as to light and shade, and therefore readily become difficult of recognition where the light-sense is altered in this way.

The colour-sense is impaired mostly for the red-green series of sensations, corresponding pretty closely, though not absolutely, to what characterises cases of congenital so-called colour-blindness (*q.v.*).

The diminution in visual acuity is an evidence of the form-sense defect, but the extent of the defect in this sense, as in that of colour, varies in different parts of the scotoma, being most marked at its mid-point.

Patients suffering from tobacco amblyopia do not complain of any pain in the eyes, and beyond such conditions of ill-health as pre-existed, and may even be assumed to have predisposed, in the way above referred to, to the poisoning, are otherwise in good health. It may be noted more particularly that there are no constant concomitant disturbances in connection with other parts of the nervous system. Curiously enough, and especially when they belong to the working classes, patients rarely present themselves for special treatment until the vision is considerably impaired, so as to amount to not more than one-eighth or one-tenth of normal. No doubt, were the affection more generally understood, it might frequently be diagnosed at a much earlier stage. Because, although sometimes the symptoms rapidly develop, the onset is usually slow, and as in many other toxic conditions the sooner the use of the cumulative poison is discontinued the more quickly are its effects removed.

There are no *evident objective signs* associated with tobacco amblyopia. The ophthalmoscopic examination is, practically speaking, negative. Some writers have described a certain degree of blurring of the margins of the optic discs indicative of neuritis. Some, again, have ascribed importance to the greater degree of pallor which the outer halves of the discs often present. In the majority of cases, however, it is difficult to satisfy oneself that the visible intra-ocular ends of the nerves show any sign of abnormality. In old-standing cases there is certainly often enough the pallor referred to; but this is by no means characteristic, and cannot be regarded as having any diagnostic importance. Recently retinal macular changes have been described as associated with the amblyopia. These, if they do exist, are sufficiently slight to escape the notice of most observers, even when they have been specially looked for.

Diagnosis.—The points by which the diagnosis of tobacco amblyopia is established are: The history of the abuse or use of tobacco in some form, the, usually only gradual, failure of sight equally in both eyes, the existence of a central scotoma readily demonstrable for red or green, the normal extent of the field of vision and functional activity of the retina in all parts not corresponding to the scotoma, and lastly the absence of ophthalmoscopic signs.

The central scotoma alone occurring in both eyes of a man of about forty years of age or older, who is at the same time a smoker, is of itself strongly suggestive of tobacco amblyopia. In much younger people it is well to make a more careful examination of the exact extent of the scotoma, and to watch particularly for subsequent ophthalmoscopic changes. There is a form of *hereditary atrophy* of the optic nerve which at first presents symptoms which might readily be mistaken for those of tobacco amblyopia.

Cases of this nature, besides occurring almost invariably before the age of thirty-five and usually about twenty, are characterised by a much greater irregularity in the shape and extent of the scotoma. The visual acuity is, moreover, generally very different in the two eyes, the blindness being more marked on the one than the other.

Finally, it is not long before very evident atrophic pallor of the discs makes its appearance.

Apart from such cases, which are not common, there are others in which a differential diagnosis may have to be made. *Retrobulbar neuritis*, for instance, may be associated with such slight ophthalmoscopic changes that, when the history leaves one in doubt, it is necessary to investigate the nature of the scotoma in either eye. The scotoma is in such cases never so clearly circumscribed as in toxic central amblyopia, and usually very different in the two eyes.

Again, in order to differentiate between the affections under consideration and certain forms of *tabetic blindness*, it is well to make a routine practice of testing the knee jerk. In case of absence of this tendon reflex, especially if combined with the suspicious appearance of the discs, it is advisable to give a guarded diagnosis, even when the history might lead one to expect tobacco blindness. It is not impossible, too, that tabetic atrophy, which occasionally begins with central scotomata, might be associated with the toxic form of amblyopia from tobacco. In cases where a doubt of this arises, the peripheral limits of the field of vision should be very carefully determined. When the cause is wholly or in part tabes, the limits are always restricted in some directions, even though only slightly.

It is also possible that cases of *chronic glaucoma* occurring in both eyes, in which the main defects are central or paracentral, might be mistaken for tobacco amblyopia. Here again, however, there are usually some ophthalmoscopic appearances which indicate the existence of something else than a true central amblyopia. The field of vision is also in glaucoma of this type, a by no means common type, restricted in some directions peripherally.

The differential diagnosis of tobacco amblyopia in all cases presenting any doubt will depend, therefore, mainly on the equality of the visual defect in the two eyes, together with the absence of the ophthalmoscopic changes.

Concomitant Conditions.—In making the subjective examination it is, however, necessary to bear in mind that certain concomitant conditions may modify the results obtained. *An inequality of defect in the two eyes* may be due to different conditions of refraction, to corneal nebulæ, to changes in the deeper part of the one eye, or to some congenital weakness, the cause of which may not be apparent, but as to the existence of which there may be a sufficiently definite history.

In testing for the scotoma with small green or red objects it may sometimes be found that a difficulty may be experienced in detecting any greater defect of vision for these colours than that which exists in the more peripheral portion of the field. This is the case where there is congenital red-green confusion, or so-called *colour blindness*, a condition which, as might be supposed from its frequency, is not so very rarely found to co-exist with tobacco amblyopia. In such cases, instead of using test objects of green or red, all that is necessary is to make use of yellow or blue ones. As, however, there is relatively less defect for these colours, the test objects used should be smaller, or less saturated, or the test with them made at a greater distance from the eye.

The scotoma can in all cases be demonstrated and mapped out by using even a white object, if the same point be attended to, viz. what is essentially the question of the size of the corresponding retinal image. This must be much smaller than that formed of a coloured object, to admit of the object being recognised in its proper colour, as in this case one is practically testing the form-sense, or acuity, and not the colour sense.

Prognosis.—In an uncomplicated case of tobacco amblyopia the prognosis, *quoad cæcitatem*, is always good. Complete blindness never takes place, although in some exceptional cases the central scotoma, which is a relative one, may become nearly absolute. The prognosis, *quoad restitutionem*, depends upon how far the patient has given up the use of tobacco, upon the length of time the poison has been acting, and upon certain conditions of general health. As a rule four to six weeks elapse after giving up tobacco before any very definite improvement begins. Sometimes even it is found that the patient seems worse for a week or two. The effect of the cumulative poison has not necessarily reached its height when treatment has been begun. After improvement has begun, however, it is usually rapid, so that in two to six weeks afterwards the vision may be practically normal, though there often remains for long some demonstrable defect in the light sense. In cases in which recovery is unusually slow it will generally be found that tobacco has not really been given up. Where there is diabetes, however, recovery may, apart from this, be slow and incomplete. Again, in very long-standing cases even abstinence may not lead to complete recovery, though some improvement may be expected in the course of time.

Treatment.—The main treatment consists in stopping tobacco. Although a diminution in the amount consumed or the substitution of a lighter quality may suffice to permit of recovery, it is always well to advise the giving up of smoking altogether. In some cases, without any change in the amount consumed, and merely as the result of some improvement in general health, the amblyopia may pass off. This, however, should not influence one in counselling abstinence. In addition, some tonic treatment is usually advisable. This should include strychnine. Strychnine appears to lessen the crave for tobacco. During the first four weeks trinitrine may be tried in doses of the $\frac{1}{200}$ gr. twice or thrice daily. Afterwards iron in some form is useful.

Lead Amblyopia and Amaurosis.—In cases of chronic lead poisoning various retinal changes are met with, accompanied with corresponding symptoms. These cases, however, do not properly come under the category of amblyopia in the sense in which it is here understood. Occasionally, however, there does seem to be some defect, mainly, too, of the central portion of the field, caused by chronic lead poisoning, which is at first altogether unaccompanied by ophthalmoscopic changes. In these cases the prognosis is bad. Further deterioration of vision, with changes in the optic nerve and retina, follow.

There is a form of amaurosis due to lead which is interesting in that, notwithstanding the complete blindness of both eyes, recovery almost invariably takes place. There can be little doubt that in such cases the blindness is caused by alteration of some kind in the visual centres. The poisoning has been supposed to be uræmic. Usually there are other marked symptoms of severe cerebral disturbances, accompanying the amaurosis.

URÆMIA, as a cause of amblyopia, or of even complete blindness, is met with either as a complication along with retinal changes, or alone. Where, in the course of an albuminuric retinitis, blindness becomes suddenly complete, the cause has to be looked for elsewhere than in the retinal changes, and is in fact a toxic one. Short of this complete blindness, however, it occasionally happens that temporary aggravations in the amount of the blindness caused by the retinitis have a uræmic origin. This is important to note both as regards the general condition of the patient and the prognosis as to vision. Uræmic amblyopia or amaurosis occurring alone and unassociated with retinitis is very rare.

B. FUNCTIONAL AND REFLEX AMBLYOPIA

TRANSITORY AMBLYOPIA.—What is often called *amblyopia fugax* is a condition which is mainly connected with gastric or hepatic disturbances. There is a transient loss of sight, either complete or partial, which comes on either quite suddenly or within a few minutes.

The duration varies from a few minutes to an hour or more, but is usually not much longer than half-an-hour. In many cases there is at the same time, or there comes on shortly afterwards, a more or less marked attack of megrim. But this may occasionally be altogether absent, or be only sometimes an accompaniment of the fugitive amblyopia in any particular individual. A peculiarity of this form of blindness is that it is *invariably negative* in its character. The patient is simply conscious of the sight becoming defective either over the whole field, or more frequently in one half or sector. He observes that while one part of an object may be quite sharply seen, other parts are either wholly invisible or only imperfectly seen, without there being any sensation as of a screen interposed. Frequently the attacks begin with the sensation of zig-zag flashes of light, arranged, it may be, like ramparts in a fortification. This peculiarity has given rise to the name teichopsia (from τεῖχος = a rampart). The blindness, whether involving the whole or only the one half of the field, either spreads from centre to periphery and disappears eventually in the same way, or the converse may take place. There are no ophthalmoscopic changes observable during an attack. *The prognosis* is invariably good, and the immediate cause is, no doubt, some vasomotor change in the visual centres.

There appears to be a form of amblyopia which is very similar in its symptoms. But the attacks are of longer duration and are accompanied by more or less *evident signs of œdema* of the retina. The cause is then a spasmodic constriction of the retinal arteries. The prognosis in such cases, though favourable, is not absolutely so. Sometimes a permanent scotoma may be left. Such cases are, however, infrequent, whereas the ordinary megrim attacks of transitory amblyopia are, comparatively speaking, frequent.

HYSTERICAL AMBLYOPIA.—Another characteristic form of functional amblyopia of quite a different nature, however, is what is frequently called *hysterical amblyopia.* Many cases have been described as *reflex*, particularly such as seem to be brought on by lesions or irritations of the fifth nerve. They are, however, all similar in nature to hysterical amblyopia. It is not necessary, therefore, to relegate them to separate categories.

The *causes* of this form of amblyopia are numerous. Amongst them may be mentioned mental shocks, neurasthenia, anæmia, and mal-nutrition generally, menstrual and ovarian troubles, frights, traumata. Railway accidents furnish a not inconsiderable number of cases. It is well to remember this, because the conditions are often such as to suggest simulation. And indeed the moral nature in hysteria is often so perverted that deceptions are frequently practised which would probably not be the case were the individual in perfect health. It is necessary, therefore, sometimes to make the somewhat subtle distinction between symptoms which are directly hysterical and those which are simulated as the result of deceptions to which the hysterical state predisposes.

Hysterical amblyopia is most common in women, though by no means confined to them. It is met with either in one or both eyes and in very varying degrees of severity. It is often accompanied by other neurasthenic or more markedly hysterical symptoms—yet this is not invariably the case. In no case which could properly be diagnosed as hysterical are there any ophthalmoscopic changes.

The different forms of hysterical blindness may be classified as follows: *bilateral hysterical amblyopia, unilateral hysterical amaurosis* and *bilateral hysterical amaurosis.*

In all cases it may be said that the symptoms seem to be due to an imagined interruption of, or interference with the establishment of, visual impressions. There exists a more or less complete cutting off of the connection between the higher centres and the peripheral stimuli. In some instances this does not amount to more than a degree of blunting of the attention. In others it seems impossible for the individual to direct attention to the impressions formed on one or both retinæ at all.

In hysterical amblyopia of the lesser degrees the result of an examination of the visual functions, of the central acuity, and of the field of vision depends very greatly on the individuality of the examiner. It depends, in fact, upon the extent to which he can stimulate and compel the attention of his patient. No doubt, also, many observers allow themselves to be deceived much more readily than others. Sometimes indeed the patient finds himself in face of an examiner whom he feels more or less powerless to deceive at all. Hence it is no doubt that the accounts given by different authors are so conflicting, and so different, it may be, from one's own experience, though that may have been equally extensive or more so.

Certain points, however, may be looked upon as fairly characteristic of the defective vision, which may be considered hysterical. In the first place the patient is usually, though not invariably, one who has from time to time shown *other nervous or hysterical symptoms.* Often, too, there are at the same time *symptoms of asthenopia* and blepharospasm or even convergent spasm. Localised hyperæsthesia or hemianæsthesia may also be present. As to vision, it is found on examination to be *more or less impaired*, often less so in semi-darkness than in ordinary daylight. The examination with the perimeter shows a *concentric limitation of the field of vision.* This limitation shows two peculiarities. It is less marked the farther the point of fixation is removed from the eye, and it varies according as the object with which the test is made is moved from the periphery towards the centre, or from the centre towards the periphery. Many explanations have been offered for these peculiarities, but there is little doubt that they depend merely upon the rapid tiring of attention. Indeed much the same may be found in the case of the normal individual. There is also often in hysterical cases a curious *perversion of the peripheral colour sense.* Blue and yellow objects are recognised as such over a smaller extent of the field than red and green. This is the reverse of what holds good in the normal state, as well as in all real diseases of the eye.

From what has already been said we may take it that although it is convenient to apply the generic term of "hysterical" to this form of amblyopia, it is by no means always, strictly speaking, hysterical. It is met with almost exclusively in young individuals from the time of puberty to the age of twenty-five or thirty. The other forms which remain to be described are truly hysterical.

Unilateral Hysterical Amaurosis.—There is nothing which, under normal conditions, can be compared to this affection. The peculiarity lies in this, that when the eye which the patient admits to be the seeing one is occluded no visual impression is received through the other, although with both eyes open it can readily be shown, by various easily applied tests, that both eyes not only see but often see equally perfectly. If, for instance, a prism of 8° or 10° be held in front of the good eye when both eyes are open the patient is at once conscious of seeing double. Of the double images, too, the one corresponding to the supposed blind eye is usually the sharper. This is because that eye alone fixes centrally.

It is not therefore a mere suppression of the one eye's images while attention is directed to the retinal images of the other. This one is familiar with, for instance, in using a microscope or telescope. The affected eye is only of use in hysterical blindness when both eyes are open to receive impressions.

Bilateral hysterical amaurosis is much rarer than unilateral. It may last for months; sometimes after passing off it may recur. In most cases the pupils are widely dilated and may not even react, or only slightly so, to light. There is apparently in such cases an entire absence of conscious vision. Yet there is usually so much vision that the individual affected is able to do many things that could only be possible by the possession of sight.

Treatment of Hysterical Blindness. — As regards the treatment of hysterical blindness it should follow the lines of treatment of hysteria generally. Suggestion of some kind must be tried, and the patients should have their interest aroused in their own case. A useful form which such treatment may take consists in the injection of distilled water under the skin of the forehead.

AMBLYOPIA DUE TO INFLAMMATION OF, OR PRESSURE ON, THE OPTIC NERVE in the orbit is a form which, though at first, it may be, unaccompanied by any ophthalmoscopic signs, always shows such signs in the course of a longer or shorter period after the onset of the symptoms. Cases of this kind will be considered under *neuritis* and *orbital diseases.*

Amblyopia, again, which is due to cerebral changes always takes the form of *hemianopia.* This symptom characterises an important group of cases, a description of which will be found in another article.

CONGENITAL AMBLYOPIA.—According to the classification here adopted, there remain only to be considered cases of congenital amblyopia. The most frequent cases of this group are the cases of *amblyopia in squinting eyes.* Partly owing to irregular optical defects, often not very readily diagnosed, and partly to defective development generally, one eye may be amblyopic, while the other is normal in acuity or nearly so. This is most common along with hypermetropia. This combination of hypermetropia and amblyopia of one eye readily conduces to the establishment of convergent strabismus. The habit soon acquired of mentally suppressing the image of the squinting eye causes the existing defect in it to be for the time being aggravated to a greater or less extent. But this increase in the defect is at once, or very shortly, recovered from as soon as the squinting eye, from any cause, such as temporary occlusion of, or accident to, the other eye, takes up the rôle of fixation. On this account one frequently finds the amblyopia of the squinting eye spoken of as *amblyopia from disuse* or *amblyopia ex anopsia.* This name originated from the idea that the defect was wholly, and not only partially, an effect of disuse. This conception has led to countless fruitless attempts to improve the vision of a congenitally amblyopic and squinting eye by temporary occlusion of the other. The results of such attempts are always, however, limited to the obtaining of a temporary recovery from the habit of suppression. But this is soon lost again, and consequently ceases to be of any service as soon as the better eye is allowed to resume fixation.

Though often referred to, there is really no such thing as amblyopia from disuse, in the proper acceptation of the term. The sight of an eye which has once been possessed of good visual acuity is never lost by disuse. It is a common experience, for instance, to operate for cataract, or for some condition caused by accident, on an eye which has consequently been useless for years—thirty or forty years or more, it may be—and yet to find that the acuity is at once regained when the optical requirements have been satisfied.

Disuse, however, in the sense of a *very early or congenital interference* with the proper development of vision, may result in amblyopia. Amblyopia from this cause is most often bilateral, and very often associated with nystagmus. It is important to remember that in order that the central vision should be sharp the optical conditions must be favourable, and the visual centres and their connections with the eye be in a normal state in the first years of life. If this should not be the case, some permanent amblyopia must result, even when the adverse conditions have been removed later on.

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Ambulance. *See* FIRST AID.

Amenorrhœa. *See* MENSTRUATION, DISORDERS OF.

Amnesia. *See* MEMORY.

Amputations. *See* OPERATIONS.

Amyloid Disease. *See* LARDACEOUS DISEASE.

Amyotrophic Lateral Sclerosis. *See* SPINAL CORD.

Anæmia.

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INTRODUCTION.—Anæmia may best be defined by its popular synonym "poorness of blood." The three main constituents of the blood are the red corpuscles, the white corpuscles or leucocytes, and the plasma. Theoretically, one might apply the term anæmia to any condition where one of the three was diminished or deteriorated, where any two of them had suffered, or where all three were less in amount than usual. It is but rarely, however, that we are in a position to estimate clinically the total amount of blood present in the body, and the methods which have been proposed for doing this are either impracticable or fallacious. In those cases, moreover, where we know from post-mortem observation that the amount of blood in the body is lessened, as for instance in pernicious anæmia, the relation of cells to fluid is usually so altered that we have no difficulty in coming to a diagnosis of anæmia on that ground alone. *The changes which occur in the blood plasma* are beyond doubt of extreme importance, but unfortunately our methods of estimating them clinically are most imperfect, except in a few cases. With regard to *leucocytes*, their number in a given quantity of blood bears no necessary relation to the amount of either red corpuscles or plasma. For instance, the blood after a hæmorrhage and that in pernicious anæmia have both a relatively watery plasma, and contain few red corpuscles, but in the former the leucocytes are practically always increased in number, in the latter always diminished. The leucocytes are most valuable aids to diagnosis of the cause of an anæmia, but changes in them do not enable us to conclude that anæmia is present.

There remain only *the red corpuscles*, and it is to changes in these that the symptoms of anæmia are mainly due, and by their diminution or deterioration that we diagnose the condition. The function of the red corpuscles is to carry hæmoglobin, the medium of respiratory interchange, and each unit of normal blood contains a definite number of corpuscles, each of which is charged with a definite amount of hæmoglobin. The unit in blood measurement is always taken as a cubic millimetre, and in that unit there should be in a healthy man 5,000,000 red corpuscles, in a healthy woman 4,500,000. In anæmic conditions there may be fewer red corpuscles to the cubic millimetre than normal, or, while the numbers remain unaltered, the individual corpuscles may contain too small a proportion of hæmoglobin. The net result in either case is that the amount of hæmoglobin in the unit of blood is lessened, and the respiratory interchange to that extent diminished also. Anæmia is therefore practically a deficiency of hæmoglobin.

SYMPTOMS.—There are many symptoms which are common, in varying degrees, to all anæmias, whatever their cause may be. The most striking is pallor of the skin and mucous membranes. There are also a feeling of weakness and lassitude, shortness of breath, palpitation on slight exertion or excitement, a tendency to syncope; whilst in chronic anæmias there may also be dyspepsia, headache, neuralgia, and in females amenorrhea. Loss of weight is not usual, but the muscularity is generally slight and the weight is kept up by the laying on of fat. Of physical signs the most important are the condition of the blood, to which we shall return, and that of the heart and vascular system. The heart is often dilated, systolic murmurs may be heard in all the areas, especially at the base of the heart, and the *bruit de diable* in the veins at the root of the neck. These auscultatory signs are of special importance as their diminution or disappearance may enable us to judge of the favourable progress of a case. By far the most important point is the condition of the blood. The pallor on which we are too much accustomed to rely, may be congenital, and depend on some peculiarity of the skin, or acquired, as is so often the case in people who have lived in the tropics, without the presence of any real anæmia. In no case, therefore, should the blood examination be neglected.

CAUSES.—In health there is a constant destruction of red corpuscles, and a constant replacing of the loss by the formation of new corpuscles in the bone-marrow and their passage into the general blood-stream. Some self-regulating mechanism, of which we have no knowledge, keeps the average number the same, and ensures that each corpuscle shall contain the proper amount of hæmoglobin. It is obvious that anæmia may result either from destruction or loss of corpuscles or hæmoglobin to such an extent that the bone-marrow is unable to compensate for the drain on the blood, or from so imperfect a formation of red corpuscles or hæmoglobin that the normal percentage of loss is not made up. The type of the former case is the anæmia that occurs after a large hæmorrhage; of the latter, chlorosis] is perhaps the best example. It is, however, extremely rare to find anæmias where both these processes are not going on together, for a blood which is impoverished by the loss of hæmoglobin cannot adequately nourish the bone-marrow, so that corpuscle and hæmoglobin formation must inevitably lag behind the normal rate. Clinically it is often impossible to say whether any given case is due more to blood-destruction or to lack of blood-formation.

The causes of anæmia are as numerous as the injuries and diseases which affect the human frame. The blood is so intimately associated with each tissue and organ that it inevitably reflects every change in them. Many of these changes are so slight that they escape our present methods, but even with the coarse means of diagnosis that we possess, much more can be learned from the blood in the most diverse diseases than most of us are in the habit of trying to ascertain. Certain diseases bear more heavily upon the blood and the blood-forming or blood-purifying organs than others, and are therefore more likely to cause anæmia. All conditions where hæmorrhage, acute or chronic, occurs will cause anæmia if the loss of blood is sufficiently great, or, which is just as important, is repeated sufficiently often. Hæmophilia, purpura, scurvy, nose-bleeding, hæmorrhoids, menorrhagia, hæmoptysis and hæmatemesis, are among the causes of many anæmias. If food insufficient in quantity or imperfect in quality is given, as in starvation; if the digestive glands cannot deal with it; if the intestine cannot absorb it, or if putrefactive processes go on in the bowel as the result of constipation or from the presence of parasites, the nourishment supplied to the blood-forming organs will be insufficient. If the general health is lowered by bad hygienic conditions, by the strain of pregnancy, or the drain of lactation, the blood soon suffers; and it is also surely affected if the circulation is impeded by cardiac or vascular disease, or aëration interfered with by disease of the lungs. In the two last conditions, however, cyanosis is very apt to occur, and this may prevent us from making a diagnosis of anæmia though it may really be present, as we are only able, unfortunately, to examine the blood in the peripheral vessels, and in these, in cyanosis, the proportion of red corpuscles to plasma is always increased.

If the liver, which plays so important a part in the metabolism of the blood-plasma, be diseased, the plasma may be so altered that the corpuscles break down more readily than they should do; if the spleen, one of whose functions is probably the destruction of effete red corpuscles, becomes increasingly active, anæmia will result. The kidney, if it be inflamed or cirrhosed, may cease to excrete material which, if retained in the blood, will destroy corpuscles or interfere with their formation.

A very large and very serious class of anæmias are due to parasites, animal or vegetable. Of the first class the ankylostoma causes repeated small intestinal hæmorrhages, the bothriocephalus poisons its host by dying and decomposing in his intestine, the malarial plasmodium passes through its cycle of development within the red corpuscles of its host, and destroys them in the process. Bacterial infections are still more common, and may cause corpuscle destruction either by the presence of organisms in the blood, by their toxins, or indirectly by the fever and the interference with the functions of organs such as the alimentary canal, the liver, and kidney, which they cause, or in chronic cases by the prolonged suppuration and consequent loss of albuminous material to which they give rise. Many organisms may cause anæmia in a still more subtle way. They give rise to leucocytosis, which makes it necessary for the bone-marrow to manufacture leucocytes instead of red corpuscles, and thus the available blood-forming space is seriously encroached upon.

The repeated ingestion of such mineral poisons as lead, arsenic, and mercury may produce anæmia, partly by their action on the alimentary canal, partly by a special action on the blood. Another very important class of anæmias is due to malignant disease, especially to carcinoma, and most of all to carcinomas of the alimentary canal. They are apt to be very severe, and in the absence of a discoverable tumour are very often mistaken for pernicious anæmia, from which indeed they can sometimes only be distinguished by careful examination of the blood.

But besides these anæmias "secondary" to other conditions, there are others for which the most careful search has as yet failed to find an adequate cause, and these are known as "primary" anæmias. They are chlorosis, pernicious anæmia, and the different forms of leucocythæmia. We have no reason to believe that these are really due to primary diseases of the blood-forming apparatus, or that such primary diseases exist, but no sufficient cause for them has yet been found or agreed upon. The cause of one anæmia, that due to bothriocephalus, which is clinically indistinguishable from pernicious anæmia, has quite lately been discovered, and we may hope that other varieties will be separated off. It is in the meantime to be borne in mind that we are not justified in regarding any anæmia as primary unless we have first exhausted every known means of ascertaining that it is not secondary to some lesion of organs or other influence. Generally the history and symptoms will help us; sometimes we can make the diagnosis only from the examination of the blood, and that is in any case the most important.

Condition of the Blood.—The state of the blood in primary anæmias is elsewhere described (see *Chlorosis, Pernicious Anæmia*). In secondary anæmia the condition of the blood depends more on the degree of anæmia than on its cause, and in order to understand the condition it is well to recall what happens after a large hæmorrhage. Many of the changes found in anæmia are really regenerative. As soon as a large quantity of blood is lost a call is at once made on the tissue-lymph, and on the bone-marrow; on the former mainly for fluid, on the latter for corpuscles. The exact physical nature of this process we need not discuss. The lymph taken into the blood is more watery than the normal plasma, so that a condition of hydræmia ensues, and this continues until the original volume of the blood is restored. It is a process of dilution, and until it is completed, a time which depends on the amount of blood lost, the proportional amount of red corpuscles and hæmoglobin is diminished. Large numbers of leucocytes are taken into the blood, partly with the lymph and from the lymphatic apparatus, partly from the bone-marrow, and their number in the blood is greatly increased (see *Leucocytosis*). The available fully-formed red corpuscles, of which there is always a certain number in reserve in the bone-marrow, are drawn into the blood, and the erythroblasts are immediately stimulated to proliferation, and form new red corpuscles rapidly, which are at once forwarded into the general circulation. If the original loss of blood has been slight, the new corpuscles may be quite up to the normal standard, but generally they are small and contain a subnormal amount of hæmoglobin. If the loss of blood is very great, the corpuscles may show the appearance of poikilocytosis—may be pear-shaped, oval, horseshoe-shaped, etc.—but even then the majority of them will be smaller than normal. The smallness of the corpuscles is probably an endeavour to make up for their diminution in number by presenting as large a respiratory surface as possible. As might be imagined, these corpuscles, formed in a hurry, are apt to degenerate rapidly, and we find that they often are polychromatophilic, that is, they tend to take up basic stains as well as acid ones. When the loss of blood has been extreme, the demand for red corpuscles is so great that some of the nucleated erythroblasts from the marrow appear in the blood, but these are practically always normoblasts, an evidence that the regeneration of the blood is proceeding on normal lines, but more energetically than usual. Megaloblasts are rarely found.

After the process of dilution is finished, by which the normal volume of the blood is restored, the number of red corpuscles rapidly increases, and more slowly the amount of hæmoglobin, and the albuminous part of the plasma is replaced.

In chronic hæmorrhagic conditions the same sequence of events occurs, but the regenerative endeavours of the marrow are constantly discouraged by fresh hæmorrhage, the nutritive fluid supplied to the erythroblasts becomes poorer in quality, and regeneration is feebler and less complete.

In secondary anæmias from other causes than hæmorrhage the same changes are found, varying in amount according to the severity of the case. In mild cases there may be nothing more than a lessened percentage of hæmoglobin with small size of the red corpuscles, but a normal number of them. In more severe cases the red corpuscles show poikilocytosis as well, and their number is diminished. It is only in very severe cases that nucleated red corpuscles are found; these are almost always normoblasts, except where the anæmia is due to intestinal parasites, when megaloblasts may appear, and may be more numerous than the normoblasts.

THE DIAGNOSIS is readily made from an examination of the blood (Hæmocytometer, Hæmoglobinometer, fresh and stained films), in conjunction with the state of the cardio-vascular and other systems. Having determined the existence of anæmia, it is frequently more difficult to say whether it be primary or secondary in origin. For this purpose the functions of the various organs previously referred to must be in turn carefully investigated. Any one of them may be at fault. Among the causal factors most frequently overlooked are tubercle and rheumatism in young subjects, and Bright's disease and maligant disease in later life. This will be further referred to in articles *Chlorosis and Pernicious Anæmia.* Much help can be got from the study of the leucocytes in individual cases (see *Leucocytosis.*)

TREATMENT.—It is of the first importance to recognise that secondary anæmia is a symptom, and that its treatment depends entirely upon the disease which causes it. Accuracy in diagnosis is essential to success in treatment. Anæmia resulting from too small an amount of proteid food must be dealt with very differently from that due to gastric ulcer or gastric cancer. Similarly with the anæmia induced by such conditions as malaria, septicæmia, Bright's disease, rheumatism, etc.

In long-standing anæmias the removal of the cause will not always effect a cure; the blood-forming organs may have become so feeble that they will not respond. In that case the lines of treatment indicated in the article Chlorosis should be followed.

Anæmia, Pernicious.

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Introductory.—"Progressive pernicious," "primary," "essential," "idiopathic" anæmia is the term employed to designate a group of severe anæmias which have almost invariably a fatal ending, in which, during life and after death, no sufficient cause for the anæmia can be found, and in which the post-mortem changes are mainly consequent on the anæmia so far as at present known.

The cases which come under this heading are not nearly so numerous as they were at first considered to be. Increased clinical and pathological investigation has made it possible to find a sufficient cause for many fatal anæmias which were at one time regarded as primary and pernicious, and improvements in therapeutics have saved many lives. At present the term "pernicious anæmia" covers a fairly well defined group of cases, with symptoms pretty constant and with a characteristic condition of the blood which seems at present to mark off this anæmia from most others. It is not to be forgotten, however, that the anæmias due to some intestinal parasites, in particular *bothriocephalus* and *ankylostoma*, are not to be distinguished, either by their symptoms or by the state of the blood, from pernicious anæmia, and there is little doubt that further advances will enable us to find the cause of other apparently pernicious anæmias, until at last we are able to assign a cause to every case.

The antecedents of patients affected with this disease are very various. Improper and insufficient food, bad hygienic conditions, hæmorrhages, infective diseases like typhoid fever, syphilis, and osteomyelitis, mental anxiety; among women, pregnancy, parturition, and lactation, and perhaps most frequently gastro-intestinal disturbances of various kinds, have all been regarded as causal factors in the production of the disease. They undoubtedly can and do produce anæmia, but why the anæmia in the great majority of cases should be slight and curable and only occasionally pernicious and fatal, we have yet to learn. The unknown factor is the really important one.

Males and females are affected with about equal frequency, and while the condition may occur at all ages it is most common in adult life, from thirty to fifty years of age.

Symptoms.—The disease is usually extremely insidious, and its onset may extend over several months, though exceptionally it may develop rapidly. The symptoms are those of an increasing anæmia, lassitude, weakness, disinclination for exertion and extreme fatigue after it, dyspnœa, palpitation, a tendency to fainting, increasing pallor, which is often of a markedly yellow tinge in the early stages, and is then apt to be confounded with jaundice, especially when it is associated, as is often the case, with loss of appetite, nausea, vomiting, and sometimes diarrhœa. Yet in spite of this the patient tends rather to increase in weight, though it is usually obvious that the gain is due more to an increase of fat than of muscle.

The patient does not usually seek advice until the disease is well established and the condition of the blood sufficiently altered to allow one to make an accurate diagnosis. The clinical picture is fairly constant.

The pallor of the skin and mucous membranes is the most striking symptom. It is usually described as being of a lemon-yellow tint, and this is true in a large proportion of cases, but there are other conditions—chlorosis, and other severe secondary anæmias—in which the yellow tint sometimes occurs, and it is not present in all cases of pernicious anæmia. The skin is often flabby and soft, and occasionally hæmorrhages are found in the later stages, and usually in the skin of the legs.

The alimentary system very often gives evidence of marked disturbance. The tongue is pale and flabby, not usually much furred; there is often anorexia, pain after meals, flatulence, and sometimes vomiting. Hydrochloric acid is often deficient or absent in the vomited material. Diarrhœa is a frequent and troublesome symptom, and often has a good deal to do with the rapid loss of strength. It may alternate with constipation. The liver may be slightly enlarged, and is sometimes tender on palpation.

The heart and vessels show the characteristic changes produced by anæmia to a marked degree. Palpitation is frequent, syncopal attacks not unusual, the pulse is rapid and its rate is at once affected by excitement or movement. In the early stages it may be full, later it is often small. The heart is dilated to a varying extent, and the usual systolic murmurs of anæmia are very audible, especially at the base of the heart and over the large vessels. The anæmic *bruit* is heard over the jugular vein. A point to be remembered in cases where transfusion is to be performed is that the arteries and veins, especially the latter, shrink enormously in this disease, and it is often extremely difficult to find a vein large enough to admit the transfusion canula. One would have expected that dyspnœa would be a common and prominent symptom, as the heart is so markedly enfeebled and the amount of hæmoglobin in the blood so small. But so long as the patient moves about quietly and does not exert himself unnecessarily there is little or no breathlessness; this is probably due to the gradual onset of the condition. In the later stages of the disease, however, dyspnœa sometimes becomes very marked. In the same way œdema of the feet and legs, though it is occasionally present, is seldom very great until towards the end of the case.

There is no characteristic change in the *respiratory apparatus*, though the acute inflammatory diseases may form serious complications, and the lungs usually become œdematous at last.

The *spleen* is not usually enlarged, nor are the *lymphatic glands*.

The blood presents by far the most important changes. It is often difficult to get a sufficient quantity for examination, and when a drop appears it is pale, very fluid, so that it tends to run off the ear or finger; often streaky, as if the corpuscles and plasma were not thoroughly mixed, and slow in coagulating. If a fresh specimen be at once examined the red corpuscles are seen to be few in number, not to form rouleaux, and usually to present the utmost diversity in size and shape. It is very characteristic of the disease, however, to find that the majority of the corpuscles, no matter how ill-shaped they may be, are larger than normal, sometimes they may even be 20 μ in diameter. In rare cases the corpuscles are not greatly deformed, but usually poikilocytosis is well marked, and the corpuscles may be pear-shaped, horseshoe-shaped, elongated, or oval—a very characteristic appearance—or they may be very small and may contain a relatively large amount of hæmoglobin. These "microcytes" were formerly regarded as characteristic of pernicious anæmia, but this is now known not to be the case. The extremities of the projections of the poikilocytes are often seen to move slightly in the fresh preparation, an appearance which has given rise to the idea that they are amœboid. There is, of course, no foundation for this view, the movements are due either to slight currents in the plasma or to osmotic changes between corpuscle and plasma. The number of *blood plates* is small, and the fibrin network is scanty and is slowly formed. Copeman believes that if a fresh preparation be laid aside, the corpuscles break up rapidly and crystals of hæmoglobin are formed with unusual rapidity.

The white corpuscles in the fresh preparation are also seen to be scanty.

Enumeration of Corpuscular Elements—Hæmoglobin.—When the *red corpuscles* are counted they are found to be greatly diminished. Cabot has shown by his tables that the average number per cubic millimetre when the patient comes under observation is 1,200,000, but as the disease progresses lower counts are very often found. The number may fall to 500,000, and in very rare cases to 200,000, or even lower. On the other hand, the corpuscles may be very much more numerous. The disease often improves under treatment or spontaneously, and during these remissions the corpuscles may rise to the normal number. In cases which remain nearly stationary for some time, as many do, the tendency is for the count to remain at about 1,000,000.

The *leucocytes* are almost always diminished in number. The average, as given by Cabot, is 3800, and this is certainly not too low a figure. They tend to fall as the case becomes worse, but there is sometimes a very considerable ante-mortem rise in number, which may be so great that the blood looks like that of a lymphatic leukæmia. In children an increase in the number of leucocytes is apt to occur in this, as in other anæmias of early life; but in the adult leucocytosis always indicates the presence of some intercurrent complication, such as would cause leucocytosis in a healthy person.

The percentage of hæmoglobin is, of course, absolutely greatly diminished, but it is often not diminished in proportion to the diminution in number of the red corpuscles; and this is *one of the principal characteristics of the blood* in this disease. For example, with 1,000,000 corpuscles to the cubic millimetre, *i.e.* one-fifth of the normal, one would expect a hæmoglobin percentage of 20, a fifth also. Were the case a secondary anæmia or a chlorosis the hæmoglobin percentage would more probably be between 10 and 15, as the corpuscles are small and poor in pigment, but in pernicious anæmia the percentage is more likely to be above 20, and may even reach 30 or more. The explanation of this is to be found in the average large size of the corpuscles, and their richness in hæmoglobin. The colour-index is then said to be high. A million corpuscles with a hæmoglobin percentage of 20 would give a colour-index of 1·0, with a percentage of 10 an index of 0·5, with one of 30 an index of 1·5. This is, of course, simply a way of expressing the amount of hæmoglobin that the average red corpuscle contains. It is when pernicious anæmias are at their worst that the colour-index is likely to be highest; when a remission is taking place the number of corpuscles increases rapidly, and the newly-formed corpuscles are apt to be small and to contain relatively little hæmoglobin; just as when the blood is restoring itself after a severe hæmorrhage. The colour-index then is normal, or usually below the normal.

Stained films.—When stained films of the blood are examined, the scantiness of the corpuscles, their large size, their deformity, and such peculiarities as polychromatophilia are of course brought out. It will be seen, too, that while some of them have a large pale centre, others show little trace of the normal biconcavity, and stain uniformly, or nearly so. More important than any of these changes is the presence of *nucleated red corpuscles*, and the kind of nucleated corpuscle which is present.

Nucleated red corpuscles are found in many severe secondary anæmias, and are most common of all in spleno-medullary leucocythæmia, but in these cases they are almost always normoblasts, or if megaloblasts are present, they are not so numerous as the normoblasts (*vide* "Blood"). In pernicious anæmia the reverse is the case. Megaloblasts may be found alone, or if normoblasts are present they are less numerous than the megaloblasts. It must not be imagined, however, that either variety is usually present in large number. It often means a tedious search, lasting for hours, and extending over several films before one can find a sufficiency of nucleated red corpuscles to be sure of the case.

In order to find enough leucocytes to make a differential count of the varieties present it may be necessary to spread some films of the blood unusually thickly, and to dry them at once with artificial heat. It will be found that there is a much larger proportion of lymphocytes present than normal, usually a little under 50 per cent of the whole number, but sometimes more. Their increase is at the expense of the polymorphonuclear neutrophiles, the other varieties are not specially altered. A few myelocytes are sometimes seen.

Micro-organisms have several times been described as being present in the blood, but they have never been cultivated nor shown to be pathogenic, and their appearance is certainly due either to accident or to an error in manipulation.

The amount of blood in the body must be greatly lessened, as anyone will realise who has seen the bloodlessness of a post-mortem examination in such cases, and as the shrinking of the vessels evidences. The amount of water is always increased, and the amount of albumen in the blood may sink even to a third of the normal, so that the whole blood, corpuscles and plasma, may contain less than the normal plasma.

Hæmorrhages of various kinds are frequent, though not usually large in amount. They may come from the mucous membranes, or may take place in the skin or in internal organs, especially in the central nervous system. They are most frequent, or most frequently traceable, in the retina, and are by some regarded as a *diagnostic sign of importance*, though they may occur in other severe anæmias. They may cause impairment or loss of vision, but do so comparatively rarely.

The urine often presents no abnormality. It is usually pale, but may sometimes become darker from the presence of urobilin or small traces of hæmatoporphyrin or other pigments. Bleeding may of course occur from the urinary tract. There is sometimes an excess of indican, and Hunter has described the presence of certain poisonous diamines—putrescin and cadaverin—which other observers have not met with. The amount of urea and of uric acid excreted is usually large. Albumen may appear but usually only in small quantity, even when the kidneys are gravely altered.

The nervous system must of course suffer, and we often find patients complaining of vertigo, tinnitus, depression, loss of memory, or sleeplessness.

Hæmorrhages into different parts of the central nervous system may produce their characteristic symptoms and even cause death, and the degenerative changes in the spinal cord may give rise to ataxia, loss of knee-jerks, and some disturbance of sensation. Much more rarely is spastic paralysis seen; and it is curious that in cases where marked degeneration in the cord is found post mortem, the spinal symptoms have not always been prominent at all. Sometimes the intellect remains unimpaired to the end, sometimes delirium or convulsions may precede a comatose condition, and not infrequently the patient becomes more and more lethargic, and slips insensibly into coma.

The temperature during the disease follows no definite course. During the greater part of it it may be normal, or even subnormal, but it is much more common to find occasional pyrexial attacks. These may be very short, but last usually for a few days, or a week or two at a time. The fever is seldom high, and the temperature-chart shows many remissions and irregularities. Sometimes a case will show a continued febrile condition for long periods, but this is not usual. The cause of the fever is not known. It may be due to increased blood-destruction, or, as Coupland suggested, to capillary hæmorrhages in the heat-regulating centres, or possibly to absorption from the alimentary tract.

COURSE AND PROGNOSIS.—The prognosis is always bad, but the course varies very greatly. Some cases pass steadily from bad to worse and die in a few weeks. Others get down to a low point, and may remain stationary, or nearly so, for months. Others again, with or without treatment, rapidly improve, remain practically well for a time, and then relapse, and this remission and relapse may be repeated several times. This tendency to remission and relapse must make us careful in pronouncing a case cured, and careful too in drawing conclusions from isolated cases as to the results of treatment.

As regards the elements of prognosis to be derived from examination of the blood, Cabot has a useful table, which I reproduce:—

Severe (rapidly fatal).	*Less severe (slower course).*
1. Extreme progressive anæmia.	1. Remissions.
2. High colour-index.	2. Normal or low colour-index.
3. Increase in size of red cells.	3. Normal-sized or small cells.
4. Degenerative changes in red cells.	4. No degenerative changes.
5. Numerous megaloblasts.	5. Numerous normoblasts.
6. Few or no normoblasts.	6. Few megaloblasts.
7. Lymphocytosis.	7. Normal percentage of polymorphonuclear cells.

Of these the most important are, in my opinion, the first, second, fifth, and last.

MORBID ANATOMY

POST-MORTEM APPEARANCES.—The pallor, œdema, petechiæ, etc., have already been described. The subcutaneous fat is usually increased in amount, and is often of a much deeper yellow than normal. The muscles are atrophied; pale, normal, or deep-red in colour in different cases. There is obviously little blood in the body, it is pale and watery, the serum sometimes stained with hæmoglobin. *The heart* is often enlarged, and the cavities dilated. The epicardial fat is large in amount, and the muscle is soft, friable, and shows marked fatty degeneration. This is not usually uniformly distributed, but occurs in patches, giving rise to the "tabby-cat striation." The blood-clots in the heart are small and light in colour, or altogether absent. The aorta and large vessels may show fatty patches, and these have been found also in the smaller vessels. The lungs are often œdematous, but show no other constant change. The *stomach* is pale, and the walls may be greatly thinned, while the rugæ are nearly obliterated, or the walls may be thickened and hard with prominent rugæ. Microscopically examined, the mucous membrane always shows signs of degeneration, differing in amount in different cases. The glands may be shorter than normal, and their epithelium shows fatty degeneration with disappearance of the oxyntic cells, and in extreme cases the glands may have disappeared altogether, and the stomach be lined simply by a flattened epithelium. Along with this epithelial degeneration there is more or less increase of connective tissue. The cirrhosis may be confined to the submucosa, or may involve the mucosa as well, and cause distortion of the remaining glands. Similar appearances are often found in both the small and large *intestines*. The villi are short, the Lieberkühnian crypts in both intestines also shortened, and with degenerative changes in their epithelium, and there are usually very few leucocytes to be found in the mucosa; the connective tissue is also often increased. *The liver* is usually large, fattily degenerated, and if the cut surface or sections be treated with ferrocyanide of potassium and hydrochloric acid, a blue coloration appears which is evidence of the presence of large quantities of iron loosely combined in the cells of the outer and middle zones of the lobules. The iron is accumulated in granular masses on the side of the cell next the bile capillaries, not next the blood capillaries. The amount of iron present in the liver is sometimes ten times the normal, a proportion which is never approached in any other anæmic condition. The *spleen* varies much in size and appearance, but shows no constant change of importance beyond the presence of an unusual amount of iron-containing pigment. The *lymphatic glands* are small, and I have several times found the mesenteric glands almost completely converted into fat. *The kidneys* are usually large and pale; the epithelium is generally fatty, and may contain a varying amount of iron. *The brain* is almost always very pale, but may show capillary hæmorrhages occasionally. In the *spinal cord* there is often marked degeneration and sclerosis in the posterior median columns, or the whole of the posterior columns, and sometimes also the posterior part of the lateral columns. These changes are generally most marked in the cervical region, and the gray matter and nerve roots are not affected. The *bone-marrow* presents very striking alterations. The fatty marrow in the centre of the long bones is replaced by red marrow, and when this is examined microscopically it is found to contain numerous poikilocytes, relatively few leucocytes, and great numbers of nucleated red corpuscles. A similar change is found in the anæmia resulting from hæmorrhage, for instance, but there the nucleated corpuscles are normoblasts; in pernicious anæmia a large proportion are megaloblasts. Ehrlich maintains that this is a reversion to the embryonic type of blood formation, and this preponderance of megaloblasts in the bone-marrow explains their presence in the blood in greater numbers than the normoblasts, and explains also the relatively large size of the red corpuscles formed from them. The changes in the marrow are of course regenerative, but the megaloblastic type is a very unsatisfactory kind of blood-regeneration, because it is much slower than the normoblastic, and because it produces large red corpuscles, whose respiratory surface is small relatively to the amount of hæmoglobin contained in them.

PATHOLOGY.—There are several theories in regard to the causation of the disease, none of which has met with complete acceptance, and all of which present certain difficulties.

(1) *That the disease is a primary anæmia*, a disturbance of the blood-forming function. This view came into vogue after the changes in the bone-marrow were discovered, but since similar changes have been found in anæmias which are undoubtedly secondary, it has ceased to be largely held. It is possible that in a small group of cases grave or fatal anæmia, though not exactly of the type we are considering, may result from the destruction of large areas of the bone-marrow by such diseases as syphilis, typhoid, osteomyelitis, and other septic processes, and a consequent failure in hæmogenesis. Such cases, however, are certainly extremely rare.

(2) *That the disease may arise out of an ordinary chlorotic anæmia*, where the capillary walls have become degenerated, and multiple minute hæmorrhages into the organs result. This is Stockman's view, but has not met with wide acceptance, as it is difficult to understand how the blood could become so completely different in type from that in chlorosis, and how the undoubted evidences of blood destruction could be explained.

(3) *That the disease is due to the presence of toxic substances in the blood,* which cause destruction of red corpuscles, that it is a chronic toxæmic hæmolysis. This view, in one or other form, is now most commonly held, and most in accordance with the pathological facts.

The *evidence for hæmolysis* is to be found in the condition of the blood, the presence of iron-containing pigment in the liver, spleen, and kidney, and the occasional excess of urobilin and other pigments in the urine.

The seat of the blood-destruction is not in the systemic vessels, or hæmoglobinuria would be much more frequent than it is, as Hunter has pointed out. It is almost certainly in the portal system, as is shown by the fact that the resulting iron is heaped up in the liver in a way unknown in any other anæmia. The spleen may be the actual seat of the destruction.

The *source of the toxic substances* is almost universally believed to be the stomach and intestine. The frequency with which gastro-intestinal disturbances precede and accompany pernicious anæmia, the actual anatomical changes already described, the good results which often follow the washing out of the stomach and bowel, the fact that the anæmia resulting from the presence of bothriocephalus in the intestine is clinically indistinguishable from pernicious anæmia, and can be cured by the expulsion of the worm, the fact that indican, the result of the putrefactive decomposition of albumen in the intestine, is often found in excess in the urine and disappears as the case improves, and that putrescin and cadaverin have also been found by Hunter in the urine,—all point to the stomach and intestine as the places where the toxin is absorbed. Its actual source is quite unknown; it may be bacterial or due to some other parasitic agency. It is difficult to say whether the anatomical changes in the alimentary canal are to be regarded as causing or caused by the anæmia. It is most easy to assume that the atrophy and cirrhosis precede the anæmia, that the resulting failure of digestion allows the unknown cause of the disease to establish itself in the canal, and that the fatty degeneration of the epithelium is secondary to the anæmia, like the fatty change in the heart-muscle and in the liver and kidney cells. It is of course obvious that the continued diminution in the digestion and absorption of food must interfere with the nutrition of the blood-forming organs.

DIAGNOSIS.—Progressive anæmia, with lemon-yellow pallor, a flabby stoutness, occasional febrile attacks, and the absence of any cause for the condition, should always suggest pernicious anæmia, but the final test is the examination of the blood. There the most important points are the reduction in number of the red corpuscles, especially if they are below 1,500,000 with a normal or increased colour-index, poikilocytosis, with increased diameter of the red corpuscles, the presence of megaloblasts, diminution in number of the leucocytes with an increased proportion of lymphocytes.

The presence of retinal hæmorrhages is not specially important. It is sometimes only by watching the course of the disease and the result of treatment that a diagnosis can be made. Care must be taken that the special prominence of individual sets of symptoms does not mislead.

The *differential diagnosis* from other anæmias is to be made on the following lines:—

Chlorosis.—The age and sex of the patient, the course of the disease, the good effect of iron, and in the blood the relatively small diminution in number of the red corpuscles, while the hæmoglobin is greatly reduced, the small size of the corpuscles and, if erythroblasts are present, the preponderance of normoblasts, make the diagnosis easy as a rule.

Anæmia after hæmorrhage.—If this is *acute* the corpuscles rarely fall below 2,000,000, if the patient survives; if it is *chronic* corpuscles *may* fall to 1,000,000 but the blood is then of the chlorotic type, and leucocytosis is present.

Anæmia from gastric or intestinal cancer without a discoverable tumour or other signs.—Usually the corpuscles do not fall below 3,000,000, while the hæmoglobin is much diminished. Where the fall is greater, the anæmia is chlorotic, and leucocytosis is usually present, or at least the proportion of polymorphonuclear cells is increased.

Other secondary anæmias.—A cause for the anæmia can usually be found, and when it is severe enough to bring the red corpuscles below 2,000,000 there is generally leucocytosis. Purpura, lead poisoning, and chronic malaria may cause severe anæmia without leucocytosis, but the type is chlorotic, and the history and clinical appearances are usually sufficient to keep the observer right.

Anæmia from intestinal parasites.—It is but seldom that it is necessary in this country to make this diagnosis, but in India, Egypt, and the West Indies ankylostomiasis is common, and the bothriocephalus is found in Switzerland, Italy, on the shores of the Baltic, and elsewhere. Here the blood gives us no help. The ova of the parasites must be looked for in the fæces, and if the condition is suspected, a course of thymol or other anthelmintic may at the same time confirm the diagnosis, and cure the anæmia.

Leucocythæmia.—It is only in children, where all anæmias are apt to be accompanied by leucocytosis and enlargement of the spleen, that difficulty is likely to arise. The larger proportion of myelocytes in the blood in leucocythæmia is important.

TREATMENT.—In this, as in other anæmias, the patient should be made to rest, physically and mentally, as much as possible, so that the work of the heart may be lightened. He should be kept in healthy and cheerful surroundings. From what has been said of the state of the alimentary canal, it is obvious that the feeding of the patient may be a very difficult problem. All indigestible food should be avoided. Meat is not usually well borne, and most cases do best on milk and light farinaceous foods, and it may be necessary to predigest these. Stimulants are often required. Great attention should be paid to the avoidance both of constipation and diarrhœa, and where there is any indication of undue putrefactive decomposition *washing out the stomach and the intestine* should be tried, and often yield good results. The enemata of water possibly act also by supplying fluid to the blood, and saline solution may be injected subcutaneously, or even intravenously for the same purpose. With the view of making as much of the blood as possible available for the circulation, massage has been used. Very conflicting opinions are held of the value of *transfusion of blood.* The corpuscles which are injected are soon destroyed, and hæmoglobinuria is often produced. The real facts of the case seem to be that transfusion of blood, when the case is getting worse, does harm; when it is improving, and there is a great demand for fluid, it does good; and it is at this period of the disease that enemata, and the subcutaneous injection of salt solution, should be tried.

Of *drugs*, by far the most generally useful is *arsenic*, which was introduced by Bramwell in the treatment of this disease. It is usually best given as Fowler's solution. Some cases respond almost at once to moderate doses, others require much larger ones; on some cases it has no effect. It is rarely necessary or advisable to give more than twenty minims three times a day, and it is most important to beware that it does not cause gastric derangement or diarrhœa. Should it seem likely to do so, the solution diluted with an equal quantity of water may be given hypodermically. We do not know with certainty how the drug acts, but from Stockman's recent research it seems probable that it stimulates the regenerative changes in the bone-marrow. Iron is not only useless, but often prejudicial. The only stage of the disease when it is of service is when the patient is recovering. It seems then, when given along with arsenic, to assist in corpuscle formation.

On the current theory of the disease, *intestinal antiseptics* should be useful. Cases are on record of rapid improvement with salol, β-naphthol, and salicylate of bismuth, and where arsenic fails it will always be well to try one of these or similar preparations in the fullest doses that the patient can stand. These drugs are often unsuccessful, because given in too small doses. With them large enemata should be given.

The internal administration of bone-marrow has been successful in the hands of some physicians, and by others has been found quite useless. My own experience is in the latter direction. Drachm doses of a glycerine extract of red marrow may be given, or some of the dried preparations in the market may be used. Its effect, if it has any, can only be due to the presence of some substance in it which stimulates the marrow to further regenerative efforts.

Patients that are apparently cured should not be lost sight of, and the relapses which are almost certain to occur should be treated as early as possible.

LITERATURE.—The following works and articles will be found useful, and are selected because they all, except Cabot's book, contain full bibliographies :—
1. CABOT.—*Clinical Examination of the Blood*, 3rd ed. London, 1898 (2nd ed. contains bibliography).—2. COUPLAND. "Pernicious Anæmia," in Allbutt's *System of Medicine*, London, 1897.—3. EHRLICH and LAZARUS. "Die Anæmie," in Nothnagel's *Specielle Pathologie und Therapie*, Vienna, 1898.—4. GRAWITZ. *Klinische Pathologie des Blutes*, Berlin, 1896.—5. VON LIMBECK. *Klinische Pathologie des Blutes*, 2nd ed. Jena 1896.

Anæsthesia and Anæsthetics.

This subject will be here considered in four sections:—

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General Physiology of Anæsthesia

INTRODUCTORY.—Means adopted to prevent pain during surgical operations are classed as general and local anæsthetics, according as they exercise a paralysing influence upon the central or the peripheral parts of the nervous system.

General anæsthetics are in most or all cases also local anæsthetics, and bodies such as cocaine, eucaine, and others employed to produce peripheral loss of sensation do as a matter of fact produce certain general and important effects upon the central nervous centres. The most obvious distinction, therefore, between general and local anæsthesia is that in the former consciousness and mentality are in abeyance, while in the latter only a small portion of the organism becomes insensitive, the processes of thought and action presisting.

GENERAL ANÆSTHETICS.—The commonly employed are gaseous, *e.g.* nitrous oxide. Others belong to the alcoholic series, and are taken by inhalation.

Of the first class, nitrous oxide is the only one in general use, given either by itself or diluted with air or oxygen.

The alcoholic include ethylic alcohol, ethylic ether, chloroform, dichloride of ethidene, the so-called "methylene," bromide of ethyl, pental, and a large number of bodies, which, although used experimentally, have never obtained sufficient credit to be included in the armamentarium of the working anæsthetist.

Among local anæsthetics should be included methyl and ethyl chloride, cocaine, eucaine β holocaine, ether spray (hydride of rhigolene).

The knowledge of the physiological action of anæsthetics is no mere academic study, its acquisition enables the practitioner to avoid failure and calamity, and carries his art beyond the unscientific boundaries of empiricism and guess-work.

Physiological action.—It has now been proved that the corpuscles are capable of conveying such gaseous bodies as nitrous oxide and carbonic dioxide to and from the nervous tissues; that anæsthetics act directly upon nerve centres, and not, as was formerly held, as mere replacers of the oxygen of the blood. A quasi-anæsthesia is produced when the supply of oxygen to the tissues is discontinued, as, for example, when nitrogen or other indifferent gas is inhaled, and narcotism follows the breathing of carbonic oxide, but such bodies are not practicable anæsthetics, and so will receive no further mention in this article.

The action of the alcoholic narcotics is somewhat similar in kind although differing widely in degree. The differences appear in part due to the haloid component, *e.g.* chloride, bromine—which appear in the heavier bodies (*e.g.* chloroform, bromide of ethyl) and which renders them less soluble in the blood, more poisonous to protoplasm, and more irritant to both voluntary and involuntary muscular fibre. This effect upon muscular tissue is of essential importance, for it probably tends to cause spasm of the involuntary muscular coverings of the arteries, and to superinduce the curious loss of elasticity in the heart muscle (loss of contractility) noted by M'William as well as the degenerative (? fatty) changes observed after death in animals subjected to prolonged chloroformisation. The oxygen which replaces the chlorine in the ether series is not an irritant, is readily soluble in the blood and acts as a paralyzant to the muscular tissue, although this effect rapidly passes off when the ether is no longer inhaled.

The inhalation of both ether and chloroform cause at first excitation, then paresis and finally paralysis of the bulbar centres. Upon inhalation being commenced a sensation of well-being and elation is experienced due to the increased vigour of the circulation, respiration grows hurried and thought becomes confused. The most unstable nervous centres, *e.g.* those higher centres which preside over thought, are the first to be affected, then the middle centres, the sensory and motor tracts, and finally, if the narcotism is pushed to its ultimate degree and the most stable or centres essential to life which control respiration and circulation pass under the influence of the anæsthetic, death occurs. There is little doubt that this action upon the nervous system is a vital one, and that the theories of deoxydation, destruction of the nerve elements, etc., are untenable, at all events, in their entirety.

The initial excitation which occurs leads to increased force of the heart-beat, with slight increase of blood pressure, the muscles contract vigorously, and while this assists venous circulation it may, by fixing the thorax, interfere with the due performance of respiration, and secondarily with that of circulation. The stage of excitement and of muscular rigidity varies widely in different persons. It is most marked in the healthy and the muscular; in alcoholics, it is more pronounced in men than in women, in women than in children. Little or none may be met with in young children, and in those who have suffered from exhausting disease. The stage is most conspicuous when slowly reached, and may, unless the anæsthetic is pushed, be very prolonged and difficult to control. There is a danger in this stage lest the struggles of the patient injure himself or the bystander, and especially lest the severe strain imposed upon the pulmonary circulation and right heart by the spasmodic contraction of the muscles of respiration, with the coincident fixation of the thorax, should lead to failure of respiration and syncope. When this spasm passes off, as it usually does in a very short time, if the anæsthetic is properly administered, the muscles gradually relax, breathing grows regular, and the pulse becomes softer and somewhat more compressible than normal. During the stage of excitement (2nd degree of Snow) the ocular globes move and the pupils dilate, but in the third degree of narcotism (Snow) under chloroform they contract and the eyes are fixed. This fixation of the globes is not as regularly met with under ether, nor can the size of the pupil under that anæsthetic be of much service as a guide to the degree of narcotism. The reflexes are gradually lost, and when the ocular reflex has disappeared all sensation has gone. Knee, ankle, and wrist clonus may develop or grow intensified in slight, but usually disappear in deep, narcosis. For rectal operations it is well to push the anæsthesia to a deeper degree, since reflex interference with respiration persists for some time when the stimulation is caused by the operation (*e.g.* dilatation of the sphincter) in this region. Many surgeons urge this fact as a reason for preferring ether to chloroform in such cases, since the deeper degree of unconsciousness is absolutely safe under the former anæsthetic. The passage of the patient beyond the third degree of anæsthesia is attended with grave risks. The gradual dilatation of the pupils, slowing and weakening of the respiration, duskiness of the mucous membranes and skin, and steady fall of blood pressure with enfeeblement of the heart-beat, all give evidence of the involvement and poisoning of those centres in the medulla oblongata which control the mechanism of respiration and circulation.

It is this degree of narcosis (4th of Snow), when the tissues are saturated with the anæsthetic, and all the vital processes are rapidly passing into abeyance, which, if unchecked, rapidly culminates in death. A convulsion marks the final (or fifth degree of narcosis), the medullary centres cease to exert any control, respiration stops, and almost immediately the circulation is stayed, and death irrevocably occurs. The sphincters relax, extrusion of fæces, urine, and semen occur in the final convulsion, the pupils are widely dilated and insensitive to light.

CHLOROFORM.—An essential difference, as pointed out above, exists between chloroform and ether. The former, being a protoplasmic poison of great power, not only produces the changes just mentioned as occurring during the procession of narcosis through its paralysing action upon the nervous system, an action which is common to it and all other general anæsthetics, but exerts a distinct and profound effect upon all the tissues with which it is brought into contact. Thus the vitality of the constituent elements of the muscular and of the nervous tissues appears from the first to become depressed, as evinced by the lessened contractibility and irritability. We have therefore, in estimating the influence of the narcotic, to consider not only the actual degree of narcotism, but also the extent of the action of the chloroform upon the tissues themselves. This is an important factor in the healthy, but vastly more so in the individual whose tissues are diseased or degenerated antecedently to their being subjected to the influence of chloroform.

If, for example, in a normal animal under chloroform in the fourth degree of narcotism, artificial respiration is commenced as soon as the natural rhythm of breathing is impaired, resuscitation may occur, but if the tissues have been subjected to the action of chloroform up to a certain point the recovery becomes impossible, as the vitality of the tissues is too much lowered. Under ether the narcotism of the respiratory centre is practically the same as that produced by alcohol, and cessation of respiration due to it is always remediable by artificial respiration. As soon as the ether vapour is expelled by this means from the organism, the tissues resume their usual tone. We are excluding, of course, secondary effects such as pulmonary œdema, condensation of vapour in the bronchi, etc.

It is then a matter of practical importance to learn what such tissue changes brought about by the chloroform are, and what they amount to in order that we may later on inquire how they may best be obviated. Let us consider muscle. It is easily seen if the nerveless portion of the frog's heart is dealt with and subjected to chloroform diluted to a degree which allows the muscle to gradually become paretic by maintaining an artificial circulation through it, it eventually stops, but as soon as the chloroform is withdrawn it resumes its beats. However, if this is repeated, in a very short time the muscle will no longer respond, even when a solution free from chloroform is allowed to flow through it; it dies. A control experiment performed with ether shows that the heart muscle can practically be arrested and made to resume work again and again almost indefinitely, that is to say, it will remain unexhausted almost as long as the heart will when perfused with circulating fluid. Next take nerve. Waller has shown that the irritability of isolated nerve treated by chloroform disappears seven times more rapidly than in the case of the same nerve treated with ether, and cannot again be revived. Further, in regard to involuntary muscular fibre, M'William found that the mammalian heart undergoes a gradual dilatation when the animal inhales chloroform, due to a loss of the resiliency of the heart muscle. When the chloroform is withdrawn the heart again resumes its normal size, but there is always a point beyond which, if chloroform is given, the heart muscle fails to respond. It then is thrown into a convulsion (delirium cordis) comparable to that general convulsion characterising the death-struggle of the fifth degree of narcotism. Without insisting too much upon the experiments of M'William, it is quite within the range of modern views upon this subject to say that most observers admit that some interstitial changes in the myocardium of the nature of a degeneration occurs under chloroform.

The tonus of the arterial muscles also become profoundly altered under anæsthesia, and far more markedly so under chloroform.

That the blood-pressure falls during anæsthesia is admitted by all. This fall is the regular sequel to the initial rise referred to above. The causation of this fall is even yet matter of dispute, but whether it is purely a peripheral one due to vaso-motor processes, whether due to cardiac weakening or the result of several causes is of less importance to practical men than is the recognition of the fact and the appreciation of the consequences arising from diminished arterial tension. The result of peripheral, arterial, and capillary dilatation with loss of tone leads to two important results, the gradual deportation of the blood from the arterial areas into the venous areas and the failure of nature's safeguards to the effect of gravity upon the hydrostatic equilibrium of the circulation. The experiments of Leonard Hill show that, given the due interaction of the heart and the peripheral resistance, posture as such can always be counteracted in the conscious individual. The erect position does not, under normal circumstances, deplete the cerebral circulation of blood, nor does the elevation of the feet permanently raise the arterial tension in the dependent areas of the circulation. When, however, chloroform has been inhaled the vaso-motor control is gradually lost, there is no longer a checked flow from the arteries to the veins, the safeguards are lost and the circulation becomes controlled by the influence of gravity. In the erect posture the brain grows anæmic, and when the head is depressed below the feet the brain vessels grow engorged. It therefore becomes a question not only of maintaining the dosage of chloroform within a safe limit as regards respiration, but also of checking the fall of blood-pressure so as to prevent interference with the normal safeguards.

There is no doubt that when the heart muscle, or the nervous tissue of the central nervous system, are inadequately supplied with oxygenated blood, as must happen at a certain point in the fall of arterial tension, that the vitality of their protoplasm is so lowered that even a small quantity of chloroform becomes a lethal one. This obtains both in anæmia of the brain and in asphyxial conditions. This danger becomes greater in persons who are anæmic at the time of the inhalation of the chloroform. Thus we see that to avoid chloroform fatalities we must recognise and avoid (1) over-strong, *i.e.* insufficiently diluted, vapours; (2) undue fall of blood-pressure, whether the result of cardiac weakness, *e.g.* acute dilatation; or paralysis of the vaso-motor centres; (3) insufficient elimination of the inhaled chloroform and deprivation of the required oxygen.

Whatever method of administration leads to one or other of such results is one fraught with danger.

The effects of insufficient dilution of chloroform vapour differ according to the strength of the vapour inhaled.

If a concentrated vapour of chloroform is presented to a patient at the commencement of the inhalation the fibres of the vagi in the respiratory tract are excited and the breath is held. Struggling commonly occurs, the glottis is closed, and the right heart fails to empty itself, the veins fill while arterial pressure falls. The lungs grow exsanguinated and the coronary arteries no longer supply the heart-muscle with nourishment. The asphyxia arising from the prolonged holding of the breath, and consequent cutting off of the oxygen supply, causes violent respiratory efforts, and several deep inspirations occur which rapidly convey a poisonous dose of chloroform to the heart muscle and nerve centres. The patient dies at once, respiration and circulation failing together.[1] One or two gasps may ensue which are the result of the incoordinated efforts of the respiratory centres to initiate respiration, but which are wholly ineffectual. These and the quivering arhythmic contraction seen in the heart-muscle, if the chest is opened in this stage, are alike useless in maintaining respiration and circulation, as the protoplasm of both muscle and nerve is destroyed. If the vapour is less concentrated the order of events is somewhat different. The chloroform in the pulmonary blood may pass through the heart and be carried into the circulation and so cause a fall of blood-pressure, and, by acting directly upon the medullary centres, destroy them. Whether or not the patient can be recovered in these cases depends upon the dose of the narcotic taken and the length of time it remains in the circulation. Finally, when an excessive dose of chloroform is taken by the inhalation of a vapour of a strength of over 5 per cent, but not sufficiently concentrated to cause spasm of the glottis, the blood-pressure falls gradually, the circulation slowly fails, and finally the respiration also ceases. This commonly occurs in the later stages of the inhalation, and is the result of inattention, as the phenomena of this form of accident are clear and patent to any one who is watching the patient. The immediate resort to artificial respiration in the last case rapidly restores circulation and eliminates the chloroform. When the heart, however, has undergone paralytic dilatation this recovery is less certain.

In the presence of burning oil or illuminating gas chloroform undergoes decomposition, a nauseating, irritating fog fills the room and excites painful chest-ache, cough, and coryza. This is due to the presence of carbonyl chloride. The inhalation of this gas has caused death in the case of those engaged in assisting at the operation, although no patient, so far, has died from it. Ether explodes and inflames if brought in contact with a light.

Upon the kidneys both chloroform and ether act as irritants. They may cause transient albuminuria, and the ether is said to lessen or even suppress the urinary flow. In pronounced renal disease, chloroform is probably safer than ether when given from an inhaler and in small amount. Glycosuria and hepatic disturbances occasionally follow the inhalation of chloroform, a condition kindred to acute yellow atrophy having been noted.

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[1] Hill, *British Medical Journal*, April 17, 1897.

Ether.—Ether being a feeble protoplasmic poison, does not injure the musculature of the heart nor produce a permanent effect upon the nervous tissue. Paralytic dilatation of the heart does not occur under ether. There is no appreciable vaso-motor disturbance or fall of blood-pressure. It is more irritating to the inflamed surface of mucous membranes than dilute chloroform vapour, and causes more secretion from their glands. It is asserted, as the result of careful observations, that the mucous râles occasionally heard after ether inhalation are the results of mucus impregnated with ether, which is aspirated from the buccal cavity into the trachea and at length into the bronchi. If this aspiration is prevented, the irritation of the vapour does no harm. The so-called "ether pneumonia"—a bronchitis leading to lobular inflammation, is probably caused by mucus so aspirated.

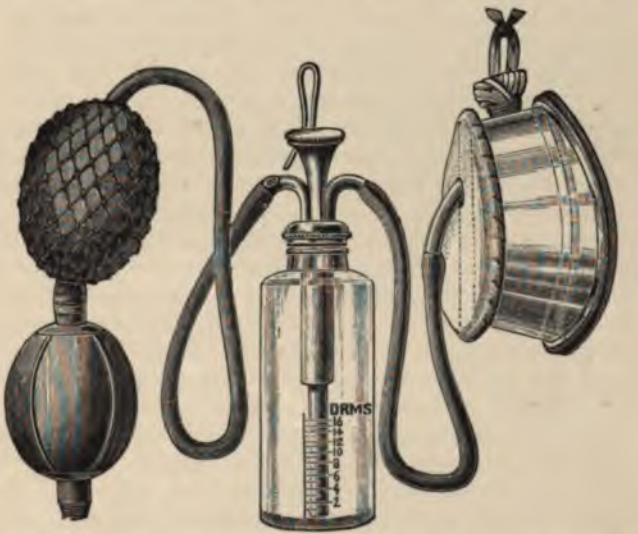
Ether pneumonia is a very rare complication of etherisation, although a kindred condition, pneumonia arising in the course of abdominal operations for the relief of obstruction, is less rare, and is sometimes confused with true "ether pneumonia." In the pneumonia following abdominal section the cause is fetid material regurgitated from the stomach and aspirated with mucus into the trachea. This danger is a well-recognised condition in patients who have not been placed under an anæsthetic.

Derangement of the mind following inhalation of anæsthetics, amounting to insanity, may occur in persons predisposed to the condition. It is probably the result of vascular disturbance and not associated with any particular anæsthetic, although it is said to be most commonly met with after the use of chloroform.

The remote effects of anæsthetics deserve careful study. During recovery, vomiting and syncopal attacks may occur, so that the recumbent posture should always be insisted upon for some hours after operation. Cases of death from asphyxia have been recorded, due to the patient having been placed in bed in such a posture that the air-passages became occluded by the tongue or by vomit. When the abdomen is very much distended the effect of anæsthesia is to relax the cardiac sphincter of the stomach so that its contents regurgitate into the buccal cavity, and, unless the greatest care is taken, become aspirated into the air-passages. This accident may take place at the time of the operation or during recovery from the anæsthetic, and may occur after any anæsthetic. It must be remembered that during the inhalation of a narcotic the body temperature falls some degrees (·5° to 3° Fahr.), the more prolonged the administration the more pronounced the drop in the temperature. Patients are thus placed in a condition favourable to the production of such inflammatory lesions as bronchitis, pneumonia, pleurisy, etc. In the case of ether the rapid evaporation from the lung surface which takes place, tends further to cool the pulmonary tissues and predisposes to such sequelæ. The patient, both during and after the operation, should be carefully covered, *e.g.* by cotton-wool, and preserved from the effects of draughts, etc.

Regulating Chloroform Inhaler.—Any plan of giving chloroform in which the dilution is only roughly ascertained by the administrator's observation is fraught with danger. The temperature of the air, the vigour of the patient's respirations, and the experience of the chloroformist are all factors in making for safety or danger. The great objection to the "open method" is that dosage cannot be scientifically determined during its use. Individual administrators may, by constant use of the "open method," acquire such judgment as will enable them to regulate the dose of chloroform with some precision; but no method should depend for accuracy upon so variable a quantity as individual guesswork. Some regulating apparatus as that designed by Junker is safer, although no inhaler can be deemed free from all danger, except in the hands of a conscientious and experienced practitioner. Undoubtedly the physical condition of the patient is an important consideration in deciding how the anæsthetic should be given. Thus in persons already placed at a disadvantage by a diseased condition of the heart muscle, by an inflamed or œdematous pulmonary mucous membrane, less of the anæsthetic is required, and much greater dilution of the vapour employed is necessary than in the case of those whose organs are normal.

The dangers of chloroform may be summarised as arising from an excessive strength of vapour, and to prevent this the apparatus figured is one of the best to use. The principle involved is that only a certain percentage strength of vapour can be inhaled. In the original apparatus of Junker this principle was carried out, but the more modern forms, such as that depicted in the figure, and those made by Krohne and Sesemann, are superior in detail. In the apparatus shown the glass face-piece (Vajna) secures cleanliness. The bottle should be capable of holding six ounces, and should be graduated to two ounces. As a rule, one ounce of chloroform, never more than two, should be placed in the bottle, being poured down the funnel attachment which has been fitted, at my suggestion, to the closing metal attachment. The afferent tube along which the entering air travels should be tipped with bone, asbestos, or some non-conducting substance to obviate freezing. The efferent tube, or that along which the chloroform-laden air passes to traverse the tube leading to the glass face-piece, must be carried down the bottle sufficiently far to prevent the chloroform entering it in a liquid state if the bottle is tilted or inverted. Care must also be taken that the tubes connected with the hand or foot bellows and face-piece



Dr. Dudley Buxton's modification of Junker's Inhaler, showing hand bellows, chloroform bottle, and glass mask fitted with metal mount, with lint stretched across.

respectively are attached to the proper taps on the metal mount of the bottle.

The bellows are at first compressed only slightly, but as the sensibility of the naso-pharynx is diminished the air may be pumped more vigorously.

In operations upon the mouth, nose, etc., in which the mask cannot be kept applied, after full anæsthesia is obtained the face-piece is quickly slipped off the tube and replaced by a soft gum elastic catheter or narrow calibred metal tube, and the chloroform given by this tube either placed in the nose (catheter) or in the mouth (metal tube).

The amount of chloroform used can by this apparatus be regulated to a nicety, and an excessive strength of vapour be completely avoided.

Any one, however expert in the use of a drop method, since "drops" are very indefinite in size, or in that of the "open method," cannot hope to secure his patient against danger in half so effectual a way as can be achieved by the use of a regulating inhaler.

As to the CHOICE of ANÆSTHETICS, the writer's experience has led him to the following conclusions:—Chloroform may be given practically to any one in whom the organs are not diseased, provided due time is allowed for the administration; a regulating apparatus is employed, and air is at no time withheld. On the other hand, for persons with disorganised circulation, especially where the heart is at fault—beating feebly, enlarged, presumably fatty; and when there is extreme shock during such operations as involve great loss of blood, chloroform is less safe than ether.

Ether may be employed for all persons and any age whose organs are normal. In the extremes of life the amount of ether given must be greatly restricted, but the youngest children will take ether well if it is properly administered. When given to children, ether should be supplemented by nitrous oxide or the A.C.E. mixture, either of these being used to induce anæsthesia and the ether employed to maintain it. Comparatively little ether is thus required. It should be given mixed with plenty of air. Those who have pulmonary diseases, habitual cough, or who are greatly cyanosed, take ether extremely badly, and it is probably safer to avoid its use in these cases and also when the kidneys are cirrhotic. The employment of nitrous oxide gas before ether obviates many of the objections commonly urged against that anæsthetic, and enables it to be employed without discomfort to the patient, without struggling, and with a much smaller expenditure of the ether than in the older method. This is an important point, since the cooling and chilling of the lungs, etc., and the "after effects" in general, are directly in proportion to the amount of the ether or other anæsthetic inhaled.

In persons who struggle much, who show a tendency to spasmodic fixation of the thorax, and who become cyanosed, the use of oxygen with ether is of great value.

At no time should ether be used by an open method.

Chloroform

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Discovery.—Chloroform was introduced in practice in the year 1847 by Sir (at that time Professor) James Young Simpson, Professor of Midwifery in the University of Edinburgh. The first persons who actually discovered its anæsthetic properties were Drs. Keith and Matthews Duncan, who were in that year, as Simpson's assistants and under his directions, testing on themselves the anæsthetic qualities of various drugs. And ever since that time, although never a decade has passed in which its reputation has not been many times assailed, it has remained the favourite anæsthetic agent of the general practitioner and of most medical men throughout the greater part of the world.

Objections to Chloroform.—A great deal can be justly said against chloroform.

For instance, while out of 6020 persons anæsthetised by sulphuric ether, or 4483 by bromethyl, only one person will die, one out of every 2286 persons chloroformed will succumb, that is—it is three times as dangerous as ether, and twice as fatal as bromethyl.

If we endeavour to kill an animal by an anæsthetic, it is easily effected by chloroform, and quickly—it can be done, for example, in 75 seconds: while with ether a similar animal cannot be killed under four a half minutes: and the relative danger of the two drugs may be estimated from this. To kill the animal with chloroform, it will be found that a small quantity, say 50 cubic centimetres of the vapour only will be needed; while, if ether be used, 500 cubic centimetres, or ten times the quantity, must be employed. If we next try to resuscitate the animals after all signs of life have disappeared, it is easily and quickly done in the etherised animal; three minutes of artificial respiration, during which 200 cubic centimetres of air have entered its lungs, will suffice to reanimate it; while in the chloroformed animal twelve minutes of artificial respiration, and a consumption of 600 cubic centimetres of air, will be called for to produce the same result.

Heart-failure.—When men or animals are subjected to the influence of chloroform, the blood-pressure is observed steadily and progressively to diminish, that is, the heart's action becomes progressively weaker as the administration goes on. Ether produces no such failure of the heart.

Syncope.—And in chloroform administration, sudden diminution, or even abolition of the blood-pressure, is often observed to occur unexpectedly at any time, particularly (*a*) at the commencement of the administration, if an additional dose of the anæsthetic be dropped on; or (*b*) while the drug is being evenly given, and the patient or animal is well under its influence; or lastly (*c*) after the administration has been discontinued and its influence is passing or has passed off. But at no moment can we be sure of the heart during the use of chloroform. Ether, on the other hand, does not show these sudden heart failures.

Chloroform a Poison.—Tested in any way, chloroform shows a poisonous action. Thus the more concentrated the mixture of chloroform and air which is employed in experiments on a series of animals, the more frequent are the fatal results during a definite duration of the anæsthesia, and these fatal results occur in exact proportion to the concentration of the mixture.

When the vapour employed preserves the same concentration throughout, in a series of experiments on animals, where the duration of the anæsthesia is the subject of study, a certain member will die under, say, half an hour of its influence, but more than double the number will die of those anæsthetised for one hour, and so on; the deaths become relatively more numerous in proportion to the length of the duration of the anæsthesia.

Even when given too diluted to cause anæsthesia, chloroform still acts as a poison, and will kill. Animals placed in an atmosphere of chloroform, so largely diluted as not to render them insensible, are so affected by it that some of them will die, and *relatively* more of them die in proportion as the time during which they are exposed to it increases. A certain number of them will waste and die even during the first week or two after they have been removed from the dilute atmosphere of chloroform.

Chloroform causes fatty or granular degeneration of the ganglionic cells of the heart, of the muscular heart fibres, of the muscle cells throughout the body, and of the cells of the kidneys, liver, abdominal glands, tunica intima of the aorta, adipose tissue, etc., and these changes may produce a fatal result days after the administration.

Such changes explain the frequency, after chloroform anæsthesia, of sickness, jaundice, albuminuria, cylindruria, wasting, debility, and death. Every experienced chloroformist or operating surgeon meets with such cases among his patients, particularly in already weakened kidneys, in hepatism, and in instances when the anæsthesia has been prolonged.

The use of ether is less frequently followed by cellular and visceral changes.

All this constitutes a very strong indictment against chloroform as an anæsthetic, and the truth and force of it must not be denied, although care and proper management can greatly lessen all of the dangers that have just been pointed out.

ADVANTAGES OF CHLOROFORM.—Chloroform is indispensable despite its drawbacks. Notwithstanding all that has been truly advanced against its use, it has always been, and to all appearance will always remain, the drug for producing anæsthesia which the general practitioner and most specialists prefer to ether and other rival agents. To them it has proved so indispensable that time and argument have failed to shake the favour with which they regard it, or the frequency with which they employ it. Even after using the others, as everyone has some time or another done, they return to the former conviction that chloroform is the best anæsthetic for general purposes. And the reasons are not far to seek.

1. *Pleasant Administration.*—Chloroform is, compared with ether, easy and pleasant in its administration. When given with care and properly handled, the worst that can be said of it in this respect is that it is unpleasant. It produces little or no choking, so that even children will often quietly submit to inhale it without resistance, and any resistance offered to it, if well given, is not due to the surface effects, but to the sensation of commencing mental confusion, throbbing in the head, and such-like feelings. But ether vapour is so irritating to the respiratory tract that it causes spasm of the glottis, and a sense of suffocation in all persons, and however administered. Even a person already under chloroform will choke and struggle violently if ether be substituted.

2. *Small supply required.*—A small quantity of chloroform is sufficient to produce anæsthesia, so that a medical practitioner, by carrying in his pocket a bottle containing four or six ounces, is in a position to anæsthetise several persons in succession, or to meet the requirements of the most prolonged operation. And he is neither stupefied himself, nor does any odour of the drug cling to him for any time afterwards.

But with ether it is otherwise: were he to trust to it, a supply of several pints of the drug would have to be carried with him to furnish him with a like power, and this quantity is out of the question in general practice, and, indeed, often elsewhere as well. For an hour or two after administering ether, some slight feeling of confusion is often experienced by the administrator, while the odour that clings to his clothes and breath for a like period is unpleasantly suggestive to bystanders of intemperate habits. In childbed, so frequent in the general practitioner's daily work, ether is, even apart from the above consideration, an unsatisfactory narcotic, and this has also its influence on his selection.

3. *Quiet Anæsthesia.*—Chloroform very rarely produces an excitement which is unmanageable, so that, single-handed, a medical man can always control any degree of it that is usually met with. Ether, on the other hand, even when—which is not readily the case—its unpleasant vapour has come to be tolerated by the patient, usually tends to produce so much excitement that two or three assistants may be required to control it. Particularly in "alcoholics" the delirium it causes is very great. One has only to witness —particularly if he be the administrator—on one or two occasions, the choking, the struggling, the excitement and shouting of the patient, and to observe the consternation these produce in a private house, to comprehend why ether is no great favourite with the general practitioner, and to share his preference of chloroform.

4. *Chloroform non-inflammable, and less volatile.*—Ether is inflammable, and its vapour is explosive at low temperatures, while you may with impunity drop a lighted match into a bottle of chloroform. And since a great number of the cases where anæsthesia has to be induced by the general practitioner occur in small rooms, at night, and in the vicinity of fires, lighted candles, lamps, or gas-jets, the properties of ether are not such as that it can, with a quiet mind, be administered under such conditions, while chloroform may.

Ether rapidly volatilises if left in a loosely stoppered bottle, while chloroform passes into vapour so slowly at ordinary temperatures when in bulk, that a bottle of it may be left open for weeks in an ordinary room, and will not be found to have appreciably lessened in volume.

5. *Ease of Administration.*—Ether is so difficult to administer efficiently, that it requires a good deal of skill and practice to anæsthetise a patient with it. Chloroform, on the contrary, can be, and often is, given by a nurse when no other person is present to assist the practitioner (and in cases of child-birth this is a point of no small consequence), if the nurse be watchful while giving it, a thing easily done.

MEANS OF LESSENING THE DISADVANTAGES OF CHLOROFORM.—To diminish the dangers of chloroform many measures have been suggested.

Two parts of it mixed with one of alcohol and three of ether, forming the well-known A.C.E. mixture, was recommended in 1864 by a Committee of the Royal Medical and Chirurgical Societies, and is sometimes used. But it is wanting in the advantages of chloroform mentioned above, and shares in the drawbacks of ether; while, owing to the different boiling-points of its ingredients, their vapours are not given off equally, and it is not possible to be aware of the proportion of chloroform that is being given, or to administer the drug evenly.

Where we are dealing with a diseased heart, or where collapse or shock are present, chloroform may be given to nearly complete anæsthesia, and ether then substituted to continue the narcosis. This method is often very comforting in anxious surgical operations.

The preliminary hypodermic administration of morphia, followed by chloroform anæsthesia, lessens the amount of the latter which is required. But the narcosis produced is seldom so good and tranquil, the stages are not so well marked, and an overdose of chloroform is more readily given; so that, in delicate operations, such as abdominal sections, the writer has had to discontinue the use of this method, as, besides the other risks and inconveniences, the recti muscles of the abdomen generally remained firm and contracted even when the anæsthesia was dangerously profound, and on the whole at least as much danger was added as was supposed to be saved by the lessened amount of chloroform employed.

The only useful preliminary drug that assists the anæsthesia is that introduced, it is believed, by Bouchut, in the case of children. It is very unpleasant, in private families, to compel little children to take chloroform as an anæsthetic; and their struggling and screaming when doing so, most trying to parents and relations, as well as to the practitioner himself, can, in suitable cases, be avoided by giving the child a teaspoonful of syrup of chloral—10 grains—an hour and a half, or two hours beforehand. The sleeping child can then be put under chloroform without being awakened, if it be gently and carefully begun. This may now and then be done in a sleeping child without using chloral, but it is exceptional.

Where the heart is diseased, Langlois and Maurange have recommended three or four centigrammes of oxysparteïn, with morphia, to be injected hypodermically a quarter of an hour before the chloroform is given. The writer generally uses in these cases ten or fifteen minims of tincture of digitalis three times a day, for three or four days beforehand.

Chloroform vapour has been administered per rectum to produce anæsthesia. It is difficult to see the advantages of this, while the objections to it are manifest.

Rival Anæsthetics.—To discuss the administration of ether, bromethyl, nitrous oxide, nitrous oxide followed by ether, Schleich's parenchymatous injections of weak saline solutions of cocaïne, ether and oxygen, local freezing, and so forth, as compared with chloroform for anæsthetic purposes, would be apart from the scope of this article. Such of them as are good, and they are not all good, are of course useful under suitable conditions. In large infirmaries, in operation theatres where a special anæsthetist is available, and in large towns where wealthy patients can suitably remunerate those who devote themselves to these forms of anæsthesia, nothing can be said against their use. Quite the reverse. But it must be clear that such methods are unsuitable in the hands of the busy general practitioner, the country doctor, in practice among the poor, in military surgery, on board ship, in the Colonies, or in remote and unpopulous districts, where chloroform is the only drug that can be advantageously employed.

Inhalers.—Inhalers of various kinds have often been recommended for chloroform administration, and for those who devote themselves specially to this, in hospitals and great cities, they are no doubt useful and beneficial. But for general use, all of them—even the simple mask laid over the mouth and nose, while the chloroform is dropped on it—are a hindrance rather than a help to the general practitioner, or to one practising away from great institutions. To carry an inhaler about so that it is always at hand when wanted; to clean it when, as it often will, it becomes soiled with blood, expectoration, or vomited matters; to preserve it where the smell of the chloroform adhering to it will not be unpleasant; these are difficulties whose repetition and accumulation render the inhalers unsuitable for general use. No inhaler is so good as a clean towel, on whose white surface the amount of the drug administered can be most accurately gauged, and which can, when soiled, be thrown aside and replaced by a fresh one, as often as may be required.

Administration of Chloroform. Preliminaries.—There is no advantage in using "pure chloroform," that is, chloroform prepared from pure alcohol. "Methylated chloroform," manufactured from methylated spirit, is as good, as pure, and as safe, and costs considerably less.

The chloroform used is best kept in a well-stoppered bottle, and in a dark cupboard; for if kept in the light, and exposed to the air, it may in time become "stale"; and when this happens, after-sickness is more likely to be troublesome.

When chloroform is given in a small room near naked lights, and for a long time, the vapour, decomposed by the flame into phosgen, hydrochloric acid, etc., is sometimes felt to be irritating to the eyes of the anæsthetist, surgeon, or assistants; for this there is no cure save an open door or other improved ventilation, but no harm ensues from it.

Preparation of Patient.—To prevent or diminish the unpleasant or dangerous effects of chloroform to our patients, either during or after the administration, it is well to have them thoroughly purged out daily, and light diet only given, for three, four, or five days before hand, and patients prepared in this way suffer much less than those who cannot be so treated. Even for facilitating the operation itself, this preparation is often of much service by preventing or diminishing sickness during it.

And for six or eight hours beforehand, in the young and strong, no food or liquid at all should be permitted. It is well to remember that even drinking water during this interval should be distinctly forbidden, as patients often think that, provided they abstain from food, water will do them no harm, whence unwelcome vomiting during the anæsthesia may ensue.

In the case of those who are feeble, a cup of beef-tea with half a slice of toasted bread may be given without harm, three hours before the anæsthetic is administered. And where heart-disease exists, half an ounce of wine or brandy diluted with the same quantity of water, if given half an hour beforehand, will be of benefit in sustaining the circulation.

In emergency cases, childbed and the like, these preparations are inpracticable, or may, if practicable, be injurious, particularly in cases of shock and hæmorrhage. Then chloroform should be given without any such preliminary preparation.

In cases of intestinal obstruction, strangulated hernia, and similar conditions, where the stomach is distended with stercoraceous or other liquid, and perhaps the abdomen also by tympanitis, it is well to empty the stomach by a stomach-tube, for suffocation by the vomited liquid is likely to occur during anæsthesia under such conditions.

The position of the patient should, if possible, be recumbent—on the back with a low pillow under the head; but this may be departed from if there be any sufficient reason, as in minor operations on the throat where roof-light is not obtainable.

The clothing of the patient should be, before the anæsthesia is commenced, so arranged that artificial respiration may, if required, be commenced without any delay. It is best to be of woollen materials. No bandages around the waist, chest, or neck, should remain. A loose shirt, or perhaps under-vest of flannel, open and loose at neck and wrists, and which can be in an instant torn down the front, is the most suitable clothing for the upper part of the body. Where the lower limbs are not the seat of operation they should be encased in warm flannel drawers and warm stockings, and enveloped besides in a blanket enclosing one or two covered hot-water bottles, whenever the operation may be a prolonged one involving much loss of heat. A warm room, 70° F., or even a table warmed by hot water, is, where it can be arranged, likely to diminish the danger to life in such exhausting operations.

Dental plates or artificial teeth should in every case be removed, as, even if firm, they may become detached and swallowed, or inhaled.

First Stage of Anæsthesia.—There are innumerable methods of giving chloroform—it would be impossible here to describe them—and there is no particularly transcendent merit about any. The method which experience has led the writer to consider best will therefore alone be described here, but it is not intended to convey by this that it is the only good plan, or even the best.

To avoid drops of the drug irritating or burning the skin of the face, some vaseline or olive oil should be rubbed over the chin, lips, and nose in adults, but not in the case of timid little children.

A towel is then spread over the face and chest. It should be a reasonably thick towel, entirely unfolded, and outspread lengthwise. A thin towel will let the chloroform spark or drop through its texture on to the face. The chloroform should not be dropped from a greater height than an inch and a half, for the same reason. The writer thinks it is best to cover the entire face with the towel, as this prevents the patient seeing those about him if he looks up when half unconscious, and he is less apt to talk, make personal remarks, scream, spit, etc., than he is if the eyes be uncovered. The fingers of the left hand are spread out beneath the towel over the mouth and nostrils, keeping the towel in good convex shape, and preventing the chloroform on the towel touching the face; while the breath can be felt on the fingers, and the facial, temporal, or carotid pulse by the thumb or little finger. By thus covering the face, a view of the expression and changes of colour, which may be helpful to a timid or inexperienced chloroformist, are excluded; but to the skilled administrator these are not things of much importance.

As the towel lies on the chest and abdomen, it serves the purpose of indicating the play of the respiration in the most sensitive manner, the light and shade on its folds forming the most delicate spirograph conceivable.

The bottle is adjusted by stopper or finger so as to yield about two drops per second, the rate being altered as required by tilting the bottle more or less. Dropping bottles are useful and are often used, but may not be always available, and it is preferable, at least for most students and practitioners, to use only things that are always at command.

Commencement of Administration.—A single drop is now let fall on the towel an inch below the point of the nose, a second drop follows in three or four seconds more, a third three or four seconds later, and so on, until the patient is accustomed to the drug, and can bear it without a sensation of choking. The patch on the towel wetted by the chloroform will remain about the same size, and so the quantity of vapour given off will be nearly constant, and the danger of heart-failure from altered concentration lessened.

When the patient has become used to it, a drop every two seconds may be given, the wet patch on the towel becoming larger, and the dose of the vapour increased. Such a dose will usually anæsthetise in a few minutes, especially if the patient breathe through the mouth, as so many do.

But if the patient, as is frequent, persistently breathes through the nose, chloroform must be given much more freely. A nose-breather requires, and can safely bear, three times as strong a vapour as a mouth-breather. Whenever, therefore, the patient does not, within two or three minutes, begin to show that he is becoming unconscious, by little purposeless voluntary movements of hands, head, or legs, or by some unconnected or semi-unconscious talking, the chloroform should be dropped on more quickly, a drop every second or even oftener, and the wet patch increased by letting the drops fall regularly all around its edges as well as in its centre. One can exactly gauge the strength of the vapour by the size of the patch, and so can easily preserve the same concentration, or increase or diminish it at will.

Stage of Excitement.—The patient will now make the movements or noises referred to, and these will continue for two or three minutes, becoming gradually more marked and then lessening as the stage of excitement passes into the next stage.

Once in a hundred cases, or so, a patient, and generally it is a female, may show no signs of excitement during the first stage. Many on the other hand, especially men, show a boisterous excitement, attempt to sit up on the table, to wrestle, fight, and so on—speaking, entreating, remonstrating, threatening, singing, praying, even cursing, the while: and such individuals are always, the writer believes, habitual alcoholics. They should be restrained, one assistant leaning over the knees, another holding a wrist in each hand, while the chloroformist keeps the head down and prevents the patient sitting up, by the pressure of his left hand upon the brow. When single-handed, the anæsthetist can best control a struggling patient by leaning with his left chest over the knees, grasping both wrists in his left hand, and continuing the administration and management of the head with his right. These alcoholics often restrain their breathing during their struggles until they become cyanosed; and in this event the chloroform should be discontinued until they have drawn a deep breath or two, re-aerated their blood, and relieved the venous congestion of the right heart. Despite what has been stated, no harm comes of firm restraint, avoiding of course the appearance or reality of roughness; on the contrary, the delirium is usually less if the restraint be firm and early applied. After a few deep breaths, the chloroform is resumed, the struggles relax, and such persons pass suddenly into deep insensibility, from which, however, they often emerge more easily and quickly than others do.

Vomiting and Retching.—In the first stage vomiting is unusual, save where the stomach is full and the patient has not been duly prepared. Should it occur, it entails much delay, but there is no help for it, the patient must be allowed, even encouraged, to vomit, and till this has been well accomplished the chloroform must be discontinued. It is the chloroformist's duty to see to it beforehand that he is provided with a small basin for such a mishap, and he ought also, by turning the face to one side and shielding it with a towel, to prevent any of the vomited matters being thrown, coughed, or sprayed on to the territory where the operation is being carried out.

It now and then happens that, without actual vomiting, strong and persistent retching takes place in this stage in persons who have not been well prepared, or even in those who have; and it is the general practice to deal with such a condition by giving the anæsthetic in increased strength—to force them past the stage of vomiting, as it were—and the practice seems to answer well. Compression of the phrenic nerves, which has also been recommended for this, is not so good, if it be of any use at all.

In goitrous patients, where there is always a certain shortness of breath from tracheal stenosis, Edmund Rose has recommended, and the advice is equally good where any respiratory abnormality is present, that the patient be allowed, without suggestion, to assume any attitude he finds best to be anæsthetised in. He is sure instinctively to select that in which his respiration is least impeded; and in this, the safest posture, he should be left undisturbed during the whole time of the administration.

Stage of Profound Anæsthesia.—In this stage, which now sets in, the limbs become relaxed by the voluntary muscular contractility becoming abolished, and immediately thereafter follows relaxation of the muscles of the tongue, throat, and, if the anæsthesia be pushed too far, eventually of the muscles supplied by the centres of respiration.

The eyeballs turn somewhat upwards beneath the upper eyelids, the iris being in a state of relaxation, so that the pupil is of medium size. In imminent death from an overdose of the anæsthetic the pupil becomes widely dilated, and when death really has irretrievably set in, it again returns to the state of medium dilatation.

Sensation next disappears successively in the skin, mucous membranes and cornea, persisting longest in this last, and in the parts about the rectum and bladder; so that the anæsthesia has to be pushed further in operations on the eyeball, bladder, and rectum, than in those on the extremities.

The pulse and respiration, generally quickened during the first stage, become again slower during that of profound anæsthesia.

During the induction of this stage, the administrator has to be careful not to give too much of the anæsthetic; and it is usual for him to test the depth of the insensibility by pinching the skin, or best of all, by touching the cornea with his finger-tip, when, if no reflex contraction is observed in the orbicularis palpebrarum, the anæsthesia is sufficient for any purpose, and should not be pushed further.

Points in Special Cases.—Certain cautions should be observed in special cases.

In minor operations, and operations on the extremities, it is often unnecessary to push the anæsthesia to its extreme degree.

Peculiarities of Children.—In the case of young children we observe, as we do in the lower animals, whose nervous organisation so much resembles theirs, that between the occurrence of profound anæsthesia and the onset of signs of imminent death the interval is a very short one compared with that of the adult, so that a few extra inhalations of chloroform may throw the child into a dangerous state. In the adult the interval is a considerable one, and the narcosis may be pushed decidedly beyond the stage of the most absolute anæsthesia without much danger. Care is therefore most important in the case of children not to continue the chloroform after the anæsthesia is complete, even by a single respiration. In children generally the anæsthesia is less regular than in adults, the cornea, for example, is often quite insensitive in them before other parts have lost their sensation.

Air-Passages—Operations.—In operations affecting the air-passages, such as cleft palate, excision of the upper jaw, etc., where blood may gravitate into the air-passages and favour inspiration of blood, infarction of the lungs, obstruction of the larynx or trachea, or subsequent septic pneumonia, it has been advised to operate with the head hanging over the end of the table (Edmund Rose's method), the blood thus running out of the nostrils and over the face, instead of into the throat and larynx; or, in lieu of this, to prevent the blood being inspired, by a preliminary tracheotomy (Trendelenburg's method), with plugging of the trachea around the introduced cannula by means of an inflatable rubber tampon or one of sponge. And in operations involving the mouth and cheeks where the ordinary administration of the anæsthetic must be arrested that the operation may proceed, inflation of the vapour of chloroform by a rubber handball or other means, has also been practised.

It is, however, far less dangerous and more satisfactory, in such cases, alternately to discontinue the chloroform entirely while the operation proceeds, and renew its administration when the patient shows signs of returning consciousness.

Far too much has been made of the danger of inhalation of blood; it is even doubtful whether, in the vast majority of cases where it occurs, it ever reaches the lungs at all, and certainly experience has shown that there is less risk to life in hazarding a little entrance of blood by using the interrupted anæsthesia just described, than in making use of either the posture of the over-hanging head, which encourages very free venous bleeding, or the method of prophylactic tracheotomy.

Falling back of the Tongue.—Falling back of the relaxed tongue during profound anæsthesia is an unpleasant and even dangerous occurrence, unless promptly treated. It occurs in many, but not all persons. When choking sounds in the throat, and cessation of the entrance of air into the chest during inspiration, show that this accident has taken place, it should be at once remedied. For this, various plans have been practised. Some place their fingers behind the angles of the jaw and press this bone forwards, relieving the obstruction by moving the base of the tongue forward with it. Others, by the utmost manual extension of the chin, raise the epiglottis from over the cavity of the larynx. But it is simpler and better to turn the face well to one side, until the cheek lies flat on the pillow, when the base of the tongue falls to one side and leaves the air-passages free.

Should this plan fail of remedying the condition, it is best to seize the tongue an inch behind its tip, between the blades of a Liston's artery forceps, one blade being placed above, the other below the tongue in its central line, and draw the organ forcibly out of the mouth. The small puncture of the tongue so made gives no trouble, and the grasp is more quickly and perfectly applied than by any of the blunt tongue-forceps invented for the purpose, and which, besides, the general practitioner is not likely to have by him.

Where it is necessary to hold the tongue forward for a long period, instead of damaging it by the prolonged or repeated use of the forceps, producing a lacerated condition that is painful and interferes with proper feeding afterwards, it is preferable to transfix the tongue in its centre, one inch behind its tip, by a handled needle, and convey a loop of strong, thick, silk thread through it, so that by passing the two free ends of the silk through the loop and drawing them tight, the organ is firmly grasped in a noose that gives a secure hold, and cuts or lacerates it less than any alternative procedure.

Apparent Chloroform Death.—Volumes have been written on the important subject of apparent chloroform death, and it has been hotly debated whether it begins by failure of the heart's action or by failure of the respiration. Both of these causes occur, but it very often, perhaps usually, commences with heart failure, at least in the more dangerous forms of apparent death. Theoretically too, differences of opinion have been expressed as to whether the circulation or the respiration alone should be watched for early evidences of danger.

But the practical chloroformist concerns himself little during the administration with such questions, and *watches both circulation and respiration* as is most natural and easy. And he observes that, when serious danger threatens, both pulse and respiration fail, and improve again together as he resuscitates his patient. Any failure of either is accompanied by an alarming change of colour of the face, and both should be treated at once and in the same way.

The operator should be warned that the patient is in danger, when he will discontinue his manipulations, make his wound as safe as circumstances permit, and leave the anæsthetist free to do what is proper.

Proposed Treatment.—As to what is proper to resuscitate the patient, opinions and recommendations have been infinitely divided. Only a brief mention of many of the proposed methods of treatment can be given here. Nélaton recommended inversion of the patient; Laborde advised seizing the tongue and drawing it rhythmically forwards twenty times per minute; inhalations of nitrite of amyl were at one time favoured; and oxygen inhalations have been beneficial where oxygen was at hand. Maas advised massage of the heart's apex 120 times per minute by the ball of the right thumb pressed on the chest wall over it; faradisation of the phrenic and pneumo-gastric nerves, galvanopuncture of the heart, acupuncture of the heart, and aspiration of the heart have all been advocated; and hypodermic injections of $\frac{1}{2}$ drachm to 1 drachm of ether, or of $\frac{1}{30}$ to $\frac{1}{12}$ of a grain of strychnia (Reid), or of 4 to 6 ounces of a warm normal saline solution into the thigh, arm, or chest wall, with friction to promote its absorption (Bobroff), have all been or are employed. And the list might be much extended.

Some of these methods have been shown to be injurious; others are not always available when required; some, although undoubtedly efficacious in certain cases, are either only occasionally or rarely called for; while others still want the test of extended experience ere a verdict regarding them can be safely given. For practitioners, therefore, they are not much to be depended on, and the duty of such a one is clearly, when serious danger threatens, not to lose valuable time in employing them, but to have recourse at once to the standard and surpassing remedy of artificial respiration, after he has made sure that the respiratory passages are free and unobstructed.

Artificial respiration has been proved not only to send air through the lungs, and so aerate the blood if asphyxia be present, supplying the best resuscitating agent to the failing nerve centres, but also in syncope to propel the blood mechanically through the heart and blood-vessels. To artificial respiration, therefore, above all other means ought the practitioner to trust, for other methods cannot replace it and are at best but doubtful adjuncts to it, and to be used only if it fails. And then they are little to be relied on.

To discuss the various ways of producing artificial respiration is not within the scope of this article.

After-Management of Cases of Chloroform Anæsthesia.—When the chloroform has been discontinued, the duties of the anæsthetist are not quite at an end.

Vomiting, shock, etc., may require his attention, and are to be dealt with on the usual principles.

The patient should not be aroused out of the slumber which generally follows anæsthesia. Nothing can be said in commendation of the practice, happily now rarely seen, of pinching the patient, shouting to him, slapping his face, or dashing cold water or wet towels on his face or chest. All such measures are uncalled for, even if not injurious.

The patient should be quietly conveyed to bed, surrounded with warm blankets and protected by hot-water bottles, to counteract the chill and lowered temperature which follow the administration of chloroform. He should be permitted to awake spontaneously after, if possible, an hour or two of quiet sleep, as he will thus be spared the initial smart of his wound, be less exposed to sickness, and the delirious talking and movements which are otherwise probable will be avoided.

It is a good rule to offer the patient neither food nor liquids of any kind until he feels a desire for and spontaneously demands them, and even then they are best given in only small quantities and of a simple nature. In abdominal sections where retching is so objectionable, total avoidance of even liquids for twenty-four hours involves no danger to the young and strong, and diminishes the risk and the discomfort.

For some days after chloroform anæsthesia, when at all prolonged, the diet should be guardedly regulated. A white tongue, a disordered stomach, nausea, sickness, bilious vomiting, and thirst, are usually observed to follow chloroform administration, and to last, gradually lessening, for three or four days. Little can be done to relieve them, save by quiet and regimen.

Inhalations of vinegar fumes from a sponge, continued for at least three hours at a time, have been said by Lewin to arrest chloroform sickness. The writer has not found them have this effect. The use of hydrocyanic acid, with warm epigastric fomentations, and where the vomited matters are acid or bilious, warm water rendered alkaline by a little bicarbonate of soda, if swallowed often but in small quantities, have seemed to him much more efficacious.

Should any signs of jaundice be observed on the skin or in the urine, the probability of some hepatic disease or degeneration is great, and should awaken anxiety, for death sometimes follows, the patient sinking from visceral changes produced by the anæsthetic.

As chloroform anæsthesia always lessens the excretion of urea for some days, the urine should be tested and watched wherever renal degeneration is suspected.

Conclusion.—The following advice regarding chloroform may be tendered. Do not fear the drug, in spite of its doubtful reputation. Prepare the patient well beforehand wherever possible. Give it boldly and evenly, but be most careful in the deep anæsthesia of children. If a mishap occurs, trust almost exclusively to artificial respiration, and use it, if necessary, for three-quarters of an hour before abandoning hope. Let the administration be as short as may be, for danger increases with its duration. And cultivate, in using it, such plans and procedures as can be employed in emergency cases, or where only the simplest adjuncts are at hand.

Ether

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IN undertaking an article on ether, I have to bear in mind three points: (1) that I am not a specialist in anæsthetics; (2) that I am, and have been for more than twenty years, an advocate of the general use of ether in preference to chloroform (of which I had previously an experience of twenty years), on account of my very firm conviction of the greater safety of ether; (3) that I am writing for the instruction of those who, not being professed anæsthetists, have more or less frequently themselves to administer an anæsthetic, or who, being operators, and having no trained anæsthetist available, have to make use of less trained persons, or even themselves to take some of the responsibility of the anæsthetic.

THE REVIVAL OF THE USE OF ETHER IN ENGLAND.—It was in the year 1873 that, having been a hospital surgeon for eight years, I heard the energetic appeal of Dr. Joy Jeffries to British ophthalmic surgeons to adopt ether instead of chloroform as a routine anæsthetic. At that time chloroform was practically our only general anæsthetic. I had witnessed two deaths from chloroform, both in slight operations, one of them, an extirpation of the eyeball in a healthy man. About the same time I had in my private practice, during a period of about two years, six cases in which I believed for several minutes the patient was dead. Two at least were dilatations of the sphincter ani, one was an operation on a child for squint, and one was passive motion for stiff joint.

Few surgeons now in practice, and fewer still of professed anæsthetists, can remember the impression made by the appeal in favour of ether by Dr. Joy Jeffries, of Boston, U.S., when at the Fourth International Ophthalmic Congress, held in London in 1873, with Professor Donders as president, Dr. Jeffries gave the opening paper of the Congress on "Ether in Ophthalmic Surgery."

From this address I quote a few sentences.

"Every week gives us some substantial proof of the fatality of chloroform in operative surgery." "I use and advocate ether, because it is as effectual as chloroform, and not dangerous to life." "That ether is more difficult to take and administer, I recognise and appreciate as well as any one. I do not wish to be hard with my medical brethren at home or abroad, but I cannot but think that the disagreeable properties of ether induce some to run the risk of the use of chloroform, which may be fatal—when, who can tell?"

I have no doubt, in my own mind, that the revival of the use of ether in England was entirely due to this crusade of Dr. Jeffries, who, during his visit, demonstrated the American method of administration at several of the London hospitals, using merely a folded towel and pouring on ether lavishly. At that time, although a few individuals were using ether, notably Mr. Warrington Hayward at St. George's Hospital, ether was practically unknown in British surgery.

At any rate the effect of this appeal upon myself and my colleagues at the Leeds Infirmary was that we at once employed ether as a duty, for a long time not exclusively, and gave it and persevered with it, in spite of the unsatisfactory methods introduced from America of the open towel, the sponge, Rendle's or Rundle's mask, and the grid (Allis's). At last Clover came to the rescue, first with his larger apparatus, July 1876, which became obsolete, and then with the smaller inhaler, January 1877 (Clover's Portable Regulating Ether Inhaler), which has pretty well held the field for administration of ether ever since. The essential point of this inhaler was the use of an india-rubber bag, into which the patient breathed over and over again. Almost simultaneously, February 1877, Ormsby, of Dublin, brought out his inhaler on the same principle, and with Clover gave the key to economy of the warmth of the exhaled breath and economy of ether, reducing thereby the risk of bronchitis and the fouling of the atmosphere of the room by the evaporation of a large amount of ether. We have to thank an American surgeon for impressing us with the conviction that ether was a safer anæsthetic than chloroform, and awakening the conscience of British surgeons to the duty of putting such a conviction to the test of experience. We have to thank Ormsby and Clover for providing us with an instrument which rescued the administration of ether from the two great evils of the American methods of administration, the chilling of the inspired air, and the great waste of ether. It was not unusual to use from six to ten ounces of ether in producing initial anæsthesia by the American methods, whereas with "Clover" one ounce or less suffices.

POINTS IN FAVOUR OF ETHER AND AGAINST CHLOROFORM.—Without going into a detailed or statistical discussion of the relative danger of chloroform and ether, I may mention certain facts which to my mind tell very strongly in favour of ether.

If we examine the *records of deaths under ether*, we find that, with very few exceptions, they occur in patients exhausted by disease, and at the fag end of life; indeed, a very large proportion of such deaths occur in abdominal obstruction where the patient is *in extremis*. The patients die, whilst under the influence of ether, from the extremity of exhaustion, not directly and purely from the anæsthetic. If we *examine records of deaths from chloroform*, what do we find? Deaths of healthy people, young and old. Deaths either before, or during, or sometimes after the termination of trivial operations, tooth-drawing, reduction of dislocation, passive motion of stiff joints, operations on the eye, and such like, deaths unquestionably due to the anæsthetic. Hewitt gives an analysis of 210 chloroform fatalities (=109 collected by the committee of the Royal Medical and Chirurgical Society, and 101 collected by Kappeler). Of these, 16 were in reduction of dislocation; 9 in examination for injuries (including putting up fractures); 16 in operations on the eye; 7 in forcible straightening of joints; 8 in application of escharotics; 7 in opening abscesses and sinuses; 18 in removal of teeth; 5 in removal of toe nail: in all 79, or more than one-third in what may be considered to be minor operations in comparatively healthy persons. Also 102, nearly half, were between the ages of sixteen and forty-five. If we turn to Hewitt's analysis of 27 collected deaths from ether, what do we there find? Twenty-four in patients suffering from "pre-existing morbid state," or from affections "producing extreme exhaustion"; 2 in moderately good health, 1 of whom died from vomited matters entering the trachea, and 1 imperfectly reported.

Take again *narrow escape from death*, alarms from suspended animation. I will only appeal to my own experience. First, the fact I have already named, that in the two or three years preceding my adoption of ether, I had in my private practice six cases of serious alarm in cases under chloroform. In the twenty years of my use of ether I have not had six such alarms. Moreover, in my casual visits to the operating theatres of the Leeds Infirmary I have, during the last six or eight years, witnessed at least six cases in which, during an operation, artificial respiration had to be employed to restore a patient who had been anæsthetised by chloroform. I have no particular recollection of witnessing any similar alarm in any case under ether. This in a hospital in which ether is much more extensively used than chloroform.

Another important point in favour of ether is this, that, after the patient is fully under the anæsthetic, consciousness of pain lags considerably behind returning mental consciousness, and this interval becomes longer towards the end of a prolonged etherisation. So much is this the case that I have known a patient converse during the insertion of stitches at the end of an operation for removal of the breast. This character of etherisation is of great value in extraction of teeth. If the ether is wisely administered and the inhalation continued three or four minutes after complete anæsthesia, so that the patient is "charged up, so to speak, with a considerable quantity of ether before the operation is commenced," a dentist may often have ten consecutive minutes for removal of teeth. This same character of etherisation is of great value in operations for cleft palate, and, in fact, when combined with the position of the head hanging down at the edge of the operating-table, it converts a difficult and fatiguing operation into a comparatively easy one.

Then *with regard to very young children*. Every surgeon will be familiar with the fact that the moment a child begins to "come round" from chloroform, it seems to come instantly and without notice to full consciousness of pain, and that often, when the child is apparently in deep chloroform sleep, the first touch of the knife will bring it back to screaming and acute sense of pain. Whereas under ether, we know when the child is anæsthetic, and we have ample notice when the anæsthesia is wearing off. For this reason I now use ether, even for infants, in a manner which I shall refer to later.

WHY HAS NOT ETHER UNIVERSALLY WON A POSITION AS THE ROUTINE ANÆSTHETIC?—Seeing, then, that for people in sound health ether is practically free from risk, whereas healthy people seem subject to as great a risk of fatal accident under chloroform as those who are worn down by disease, how comes it that ether has not universally won its position as the routine anæsthetic, chloroform being reserved for exceptional cases in which special reasons justify its exceptional use?

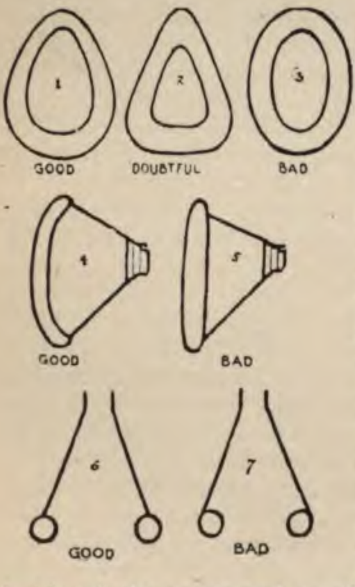
It is my belief that five main factors are responsible for this state of affairs.

1. That a bulky apparatus is needed.
2. That modern Clover's ether machines are often faulty in construction.
3. That the satisfactory administration of ether "tuto, cito, et jucunde," is a highly-skilled art, and needs to be taught and learned.
4. That ether is supposed to be extremely unpleasant to the patient.
5. That ether is supposed to be frequently responsible for fatal bronchitis, more so than chloroform, and that the deaths from this source are an adequate set-off to the very frequent immediate deaths from chloroform in healthy persons undergoing minor operations.

Factor 1. *The need of a bulky apparatus.*—This difficulty may arise in country practice, especially where the medical man does most of his work on horseback, but it ought not to influence a practitioner in a town. Indeed, as far as it is a pardonable factor, it covers a very small proportion of the cases in which an anæsthetic is required.

Factor 2. *Faulty construction of Ether Inhalers.*—That many modern "Clover's inhalers" are defective in construction, I am perfectly certain, and I am not less convinced that such defects are a great discouragement to those wishing to acquire the art of administering ether. I rarely see an inhaler which equals my own, bought 22 years ago, when first invented by Clover. Competition for cheapness, wholesale manufacture, and "made in Germany" are greatly responsible for this deterioration.

Probably the most common and the most mischievous fault of all is a *bad shape of face-piece.* There are three directions in which the face-piece may be faulty—(*a*) The Shape of the Mask, at the edge of which the hollow cushion is fixed, is sometimes oval (Fig. 3) and sometimes triangular (Fig. 2). These forms are bad. The proper shape is that of a horse-collar (Fig. 1). Seen in profile the cushioned edge should not be straight but should recede in a slight curve at the top and the bottom (Fig. 4). This provides for the projection of the bridge of the nose and of the chin, and thus allows the inflated sides to fit into the hollows of the cheeks. (*b*) The Hollow Cushion should be of soft thin rubber, thin enough to partially collapse when the air is let out, otherwise when distended with air the cushion will be little better than solid rubber, and will fail to adapt itself to the irregular contour of the face. (*c*) Another important point in reference to the cushion is that it should be so attached to the edge of the mask that when compressed against the face it should overlap both the outer and inner edge of the mask (Fig. 5), *i.e.* when in use it should not roll to the inside of the mask and so allow the hard edge to press against any part of the face (Fig. 6).



There *must be invariably two face-pieces*, one for adults, and a smaller one for younger people. For the sake of cheapness and competition, some makers issue inhalers with one face-piece only. This is a fraud, and worse, as it renders the use of the inhaler impossible for young people.

The "*whistle*" when attached to the "face-piece" should be fixed by means of the indicator which it carries into a *bayonet joint*, otherwise it is apt to become loose and slip out of its socket.

The *india-rubber bag* should be of soft rubber—and not less than 13 inches in length. These bags are often too small, and so entail more labour than need be in inhalation.

FACTOR 3. *That the satisfactory administration of ether is a highly skilled art and needs to be taught and learned.*—Unless the administrator has acquired the art he cannot give ether with comfort to the patient, or with efficiency, or with any satisfaction to himself, and he naturally falls back upon chloroform, the administration of which is more simple and apparently more easy.

Further, I know, from the experience of a long series of intelligent assistants, that the proper use of ether can be taught, and quickly taught, by one experienced in the art, by observation of a few simple rules, aided by trained and sympathetic clinical observation in the pupil.

As a formula usefully expressing in diagrammatic form the principle on which the inhalation of ether should be regulated, I often appeal to the curve of "harmonic progression," illustrated by the strings of a harp. If this curve be divided into three portions, the first third represents the period of overcoming the patient's fear, his shrinking from the contact of the face-piece, the breathing through the ether chamber, the tasting of the ether, and the inflation of the bag, each of them formidable to a timid person unaccustomed to take an anæsthetic or retaining an unpleasant recollection of a previous administration. During this period the patient's confidence is gained, and toleration of the taste and pungency of the ether vapour becomes established. The index of the inhaler would stand at the end of the period at a half. In the middle third the patient loses consciousness of the unpleasantness and choking effect of the ether and becomes mentally confused and practically unconscious. The index would have now reached one and a half.

In the third period, the patient having lost consciousness, the index is moved on more rapidly, a short stage at each inspiration, until the patient becomes anæsthetic and is ready for the operation. The index will now be at three in most cases, in others at four.

It is important that the patient should "give way" to the anæsthetic, not resist it. Some persons giving ether upset the patient at the beginning by turning on the index too rapidly without winning the tolerance proper to the first stage. Others waste time by not watching their opportunity and pushing on rapidly at the proper time. The guiding principle should be *not to push the ether more rapidly than the patient tolerates, but at the same time to push on as rapidly as you can.*

It is well to move on by very short stages of the index, especially in the early stages. Hewitt says, "As a general rule about one-eighth of an inch of the circumference of the reservoir may be removed away from the indicator every breath or two." For convenience I have spoken above of moving the index (or indicator), whereas the reservoir revolves and the index is fixed.

FACTOR 4. *That ether is unpleasant, and chloroform the reverse.*—This no doubt is true. But, if ether is skilfully administered, with the majority of people the unpleasantness will not be great nor of long duration, and with some, and I speak from my own experience in my own case, will be entirely absent.

Again, granted that ether is in many instances more unpleasant than chloroform, is this an adequate set-off against the risk?

FACTOR 5. *That ether is supposed to be frequently responsible for bronchitis more so than chloroform.*—One of the strongest points which defenders of chloroform urge as an objection to ether is the risk of fatal bronchitis. On this I have four remarks to make. (1) That if ether be properly administered, fatal bronchitis is an extremely rare event. (2) That even chloroform has been followed by a fatal bronchitis. (3) That the possibility of such a risk calls for the greatest vigilance against chilling of the patient by all who are responsible for operations in hospitals. (4) That during my more than twenty years of almost exclusive use of ether in private surgical work, I can only recall one case of fatal broncho-pneumonia as a sequel to an operation under ether. Hewitt has some significant and interesting remarks on this point, which may be summed up in this sentence, "When ether is properly administered to appropriate cases, the danger of such after-results is infinitesimal."

GENERAL RULES FOR THE ADMINISTRATION OF ETHER.—RULE 1. *As to food.* If possible the stomach should be empty. It is best to operate early, at 8.30 or 9 A.M., before any food has been taken. If the patient be feeble, or if the operation has to be later than 9, a cup of tea, with cream and not milk, should be given early, at 7. When the operation is at mid-day or later, no food should be given for four or five hours (some anæsthetists say five or six hours), before the operation, and any food so given should not be either meat or milk or egg.

RULE 2. Make sure that *false teeth* are removed before commencing to administer the anæsthetic.

RULE 3. *Have at hand tongue forceps*, or artery forceps, and a Mason's gag.

RULE 4. *See to the patient's warmth*, especially if he has to be removed from his bed for the operation. For perineal operations, it is well, besides stockings, to provide warm leggings. The leggings may very readily be made from a pair of child's cot blankets, doubled and united along the edge.

In hospitals the warmth of the patient is more difficult to secure than in private practice, and needs in proportion the greater vigilance. At the best there must be considerable loss of warmth in removal from bed to ambulance, in the journey along corridors, in the transfer from ambulance to operating-table, in exposure of a large surface of the body on the operating-table, in removal back to ambulance, back through corridors and into bed, which can hardly, even with a hot bottle, be as warm as when it was left. May not this too little estimated loss of heat be responsible for some of the bronchitis laid at the door of ether and chloroform?

RULE 5. *Approach the patient so as to win confidence*, and not to inspire or increase an existing dread, keeping the inhaler as little in evidence as possible, or even out of sight, until the patient's attention has become engrossed by the application of the face-piece. This is all the more necessary with nervous or very young patients.

RULE 6. *First apply the face-piece with the "whistle" fixed into it*, and see that it fits comfortably, is of suitable size, and by its adaptability to the bridge of the nose, cheeks, and chin, compels all respiration to pass through the "whistle."

RULE 7. *As soon as the patient has become used to the contact* of the mouth-piece and breathes calmly, remove it, join it to the reservoir, blow through the reservoir so as to get rid of any ether vapour, and then re-apply quickly with the indicator at 0. *N.B.*—If the inhaler be a new one make sure that the lettering is correct, and that when the indicator points to 0, you are not unwittingly turning on full ether. At this period the patient will taste the diffused vapour of ether, but will not inhale it, as the breath passes directly through the centre of the reservoir without traversing the ether chamber. I always use methylated ether, sp. gr. ·720.

RULE 8. *After two or three breathings through the reservoir the india-rubber bag is filled and added,* and as soon as the patient fills the bag calmly at each respiration the reservoir is turned, perhaps a fourth of the way towards 1, every third or fourth respiration. It is this period which tests the tact and judgment and patience of the administrator, as the patient is in full possession of his faculties, of his perception of the pungency of the ether, and of his consciousness that he is about to lose consciousness under the influence of the anæsthetic. Partly by control of word of mouth of the administrator, partly by the incipient effect of the ether, the patient drifts on until at the end of the "first period," and with the index at ½ or at 1, he has become indifferent to the unpleasantness of the anæsthetic process. It is at this stage chiefly that the ether vapour may cause coughing. If this happens it is well to turn back a short stage, and then after three or four breathings to go forwards again, when it will generally be found that the additional ether inhaled at the lower point has induced a tolerance which enables the patient to go to the higher point without further coughing.

During this period of administration it is important as a general rule not to remove the inhaler nor to allow the patient to breathe fresh air, but rather to encourage him to endure the transient discomfort, as it will soon pass into oblivion. Presently the patient gradually loses consciousness, breathes more quickly and deeply, and then falls into anæsthetic stertor and complete unconsciousness.

This condition may sometimes be reached in a minute and a half—frequently in three minutes, before allowing the patient to breathe fresh air, and is often accompanied by moderate duskiness or lividity. "No attention should be paid, as a general rule, to the duskiness until stertor has commenced" (Hewitt). The inhaler may now be removed for one or two inhalations, which restore the colour to the lips, and then re-applied—the index being turned back to 2 or even to 1, which as a rule suffices for maintaining the anæsthetic sleep, with considerable intervals of breathing without the inhaler. It is a mistake, made not infrequently by beginners, to retain the index at F (full) or even at 3, in order to continue the anæsthetisation after the patient has fully succumbed to the anæsthetic. The patient is thereby saturated with more ether than is needed for the purpose of the operation, and is brought nearer to the margin of safety.

After replenishing the ether go back to 1; as, in consequence of the replenishment, there is at first a very rapid evaporation of ether, and then rather rapidly move on to 2 or 3.

Mr. Walter Thompson, senior anæsthetist to the Leeds Infirmary, tells me that he hardly ever goes beyond 2. This entails a rather long period before anæsthesia, but it is a great safeguard against the collection of mucus in the throat. Mr. Thompson also prefers to give ether with the patient on the side, and the head slightly thrown back so that the saliva may run out at the side of the cheek and not collect at the back of the throat.

After the patient has fully succumbed to the ether, about ʒj every quarter of an hour should suffice for the continuance of the anæsthesia.

HOW TO DEAL WITH STRUGGLING AND EXCITEMENT.—Persons who object to ether as an anæsthetic make much of struggling as an objection. As a matter of fact, if ether is properly administered, struggling and excitement ought to be a very rare occurrence, and when they do occur they will test the tact and experience of the administrator.

STRUGGLING UNDER ETHER is generally met with in strong men, and especially men accustomed to take alcohol freely. There are, in my judgment, two distinct periods when excitement occurs, which call for distinctly different management, an earlier one, and a later one. The earlier period comes on when a patient is just losing self-control, is becoming blue, is still sufficiently conscious to feel an intensely distressing sense of suffocation, and struggles, two-thirds consciously, and one-third unconsciously, for fresh air and free breathing. This excitement is in a great measure the result of carbonisation, often combined with too strong ether vapour. The index of the inhaler will have probably reached 1½. I am quite clear as to the best way to deal with this condition. "Reculer pour le mieux sauter." Remove the inhaler, let the patient breathe freely and get rid of the carbonisation, and become calm and reassured, and then begin again at 0. Then push on, a small stage at each breath, and rapidly reach 1½ again, when it will in most instances be found that the excitement does not recur, because he now has the cumulative effect of the first and second dose of the ether. But in some few instances on reaching this point again, or further on at 2, the excitement recurs. The patient now will be more saturated with ether, will have quite lost self-control, and will not be consciously struggling for breath. The course to take now, is to slightly restrain the patient's hands so that they do not interfere with the anæsthetist, and quickly, breath by breath, to move the index up to F, when the ether asserts its control and the patient rapidly becomes quiescent, being overpowered by the ether rather than by the asphyxia.

DUSKINESS, LIVIDITY, AND CARBONISATION.—The method of administration of ether by Clover's or Ormsby's inhaler, means this, that the patient breathes the same air over and over again, perhaps for several minutes, and in time is anæsthetised mainly by the ether, and partly perhaps by the carbonisation of the blood, as indicated by duskiness or even blueness of the lips. The carbonisation accelerates the effect of the ether, and is easily dissipated by two or three breathings of fresh air, when the anæsthesia can be easily maintained by a moderate amount of ether (index at 2 or even 1), frequently intermitted without again inducing carbonisation and blueness of lips. If the patient is kept constantly carbonised, mucus is poured out into the bronchi, a condition which should rarely occur, and whereby probably a greater risk of bronchitis is incurred.

There are, however, persons, and these cases are rare, in whom some condition, probably of heart, possibly of lung, produces dyspnœa with blueness, even with the greatest care, a condition which prevails during the whole period of anæsthetisation. Such cases demand anxious watchfulness. As a rule, when there is blueness of lips the inhaler should be removed until the red colour is re-established.

ARREST OF RESPIRATION.—According to the observation of those who still advocate chloroform as the best anæsthetic, arrest of respiration is a danger-signal not less menacing than failure of pulse, and indeed is by some considered to be the one point to be anxiously watched. This sometimes leads those, who without full experience are administering ether, to take alarm when a patient in the early stage of etherisation for a time ceases to breathe. The cases, however, are very different. Under chloroform, arrest of respiration is a sign of danger, as it is caused by the poisonous effect of the drug upon the respiratory centre in the brain, and is rapidly followed by failing pulse. Whereas cessation to breathe under ether, is either a half voluntary holding of the breath which passes off in a minute or so, or is a spasmodic closing of the jaws and drawing back of the tongue, which, as soon as a certain degree of carbonisation takes place, becomes relaxed and allows normal respiration to be restored. Meantime the pulse is not failing but beating strongly. On this point Hewitt says, "The all-important point concerning respiratory failure in moderately healthy people under ether is this, '*however such failure may arise, the circulation of the patient at the moment when breathing ceases is sufficiently satisfactory for remedial measures to be almost invariably successful.*'"

After-sickness.—Mr. Walter Thompson tells me that, from constant enquiries that he has made of the ward Sisters, sickness after ether is less frequent, and much less distressing and serious, than after chloroform. This entirely agrees with my experience in private practice.

USE OF CHLOROFORM ALONG WITH ETHER.—Though I never use the A.C.E. mixture, which I believe to be only less dangerous than chloroform, in a few instances I use chloroform, chiefly as an introduction to ether, very rarely and exceptionally as a sequence.

As a preliminary to ether I consider chloroform to be useful, and justifiable, in the following cases:—(1) In very young children, to overcome fear. (2) In very timid, excessively sensitive, almost hysterical persons, who rebel against the face-piece and the smell of ether. (3) In persons who have a very distressing recollection of a previous administration of ether. (4) In persons subject to asthma and bronchitis. In such cases I give chloroform on the folded towel as I have seen it used in Edinburgh, *just up to the point of semi-consciousness when the ether can be put on without inducing distress or resistance.* Meantime free respiration is being maintained. As a sequence to ether, I have used chloroform in hare-lip and operations about the mouth and face, where, during an operation, the face-piece of the ether-inhaler could not be applied. In these cases I take care that the patient is "charged up with ether," "has been breathing ether vapour for some minutes," before commencing the operation. "When ether has been given say for 5 or 6 minutes, the quantity of chloroform then required to allay any symptoms of coming round will be very small" (Hewitt).

There is one use of chloroform after ether which I believe to be excessively dangerous, and has I believe twice led to a fatal result in the Leeds Infirmary during recent years. When a patient is violently struggling against ether, and the anæsthetist failing to induce anæsthetic sleep, attempts to conquer the struggles and resistance by chloroform. In such cases, assuming that to proceed with ether is out of the question, the only safe way probably is, to allow the patient to come round from the ether, and then to begin cautiously with chloroform.

NITROUS OXIDE AS A PRELIMINARY TO ETHER.—This subject is considered on pages 208-210.

ON ETHERISATION OF THE VERY YOUNG AND THE VERY OLD.—In discussions on anæsthetics it is not uncommon for even strong advocates of ether to make an exception in the case of the very young and the very old. I do not in my own practice, as a matter of course, except even the very old, but deal with each case on its own merits. If an aged person showed a respiratory intolerance of ether I should not hesitate to substitute chloroform, but, after the establishment of anæsthesia, I might then use ether for its continuance. But with the very young I have recently almost invariably used ether, preceded for a very short time by chloroform to obviate fear, in the following manner:—Chloroform on a handkerchief or folded towel is given until the child is just "fuddled" and will not be conscious of the change to ether. Ether is given in a small Ormsby's inhaler with the valve closed, and the effect of ether is rapidly induced. The face-piece is a round hollow pad of larger calibre than the pad of the Clover's face-piece, which easily fits either an infant or a child of one or two years of age. For such the ordinary small Clover's face-piece is too large.

ETHER AND RENAL DISEASE.—Hewitt says: "When advanced disease of kidneys is present, chloroform is said to be preferable to ether. But from the satisfactory manner in which patients with all forms of visceral disease pass through prolonged administrations of the latter anæsthetic, it is questionable whether we are justified in altogether withholding ether, at all events in some form or other, because Bright's disease exists."

ETHER IN ASTHMA AND BRONCHITIS. — Probably in most cases, and inevitably in some, the wisest course is to give chloroform for the early stage of anæsthesia, and then when tolerance of the anæsthetic has been attained to carefully introduce the ether, and if it were then tolerably borne to continue it to the end of the operation.

"ETHER IN POSTNASAL ADENOIDS, ENLARGED TONSILS, AND NASAL STENOSIS."—I have never used any anæsthetic but ether in these operations, and have never met with any difficulty or alarm, and I have been operating on such cases on the modern plan for nearly fifteen years. Owing to obstruction of the nares, it is in the first instance more difficult to get the patient well under the anæsthetic, but when the patient is once under the influence of ether there is in my experience no further difficulty, even if a reapplication of the face-piece should be needed. In these operations I invariably have the patient's head hanging over the edge of the table. The first step is to remove the tonsils with the guillotine, and the *second step is to dilate the nostrils with Hegar's dilators* up to 7, 8, 9 or 10, according to the age of the patient, and then all the rest, scraping away of adenoids, dealing with bleeding, which profuse at first before long abates, easily follow.

Apparently, if we may judge from published records, chloroform is in these cases exceptionally dangerous.

ABDOMINAL OPERATIONS.—In these I invariably use ether, and I cannot say that I have met with the difficulties of muscular rigidity which others speak of. Dr. Hewitt says on this subject, p. 49, "But the behaviour of the patient under ether is greatly dependent upon the manner in which the anæsthetic is administered."

ETHER IN OBSTETRICS.—Of this I have but little experience beyond one case. A relative had chloroform for four confinements, and subsequently of her own choice ether for four confinements. I gave the anæsthetic on most of the occasions. The point in reference to ether that struck me was this. After the patient had once been anæsthetised, if at the first indication of a pain coming on the Clover was applied, three or four deep inspirations sufficed to produce the necessary analgesia. The process was easy to manage, and very little ether was used, and the patient had no desire to return to the use of chloroform.

"CAN RULES FOR ADMINISTRATION OF ETHER BE FORMULATED?"—In the earlier part of this article, I have put into form a series of general rules for the administration of ether which I have found useful for the guidance of myself and others whom I have influenced in this respect.

It is most satisfactory and interesting to find that they are identical in principle and almost in detail with those originally set forth in one of the earliest papers written on the subject, by Mr. Cautley Dawson[1] in the year

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[1] *Brit. Med. Journal*, 2nd March 1878, p. 289.

1878, who was then my assistant, and had gained his experience in giving ether in my private practice. The same may be said of the rules set forth by Hewett in his work from which, by his permission, I have made several quotations, and by Dudley Buxton in his work on "Anæsthetics" 1888. The same may be said of the views expressed by E. F. White, one of the anæsthetists of St. Thomas's Hospital, in his speech at the meeting of the Society of Anæsthetists, 16th December 1898.[1] They are very fully reported in the *Clinical Journal* for 7th and 14th December 1898, and 11th and 18th January, 8th and 15th February 1899.

Other Anæsthetics

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The *A.C.E. Mixture*, devised by the late Dr. George Harley, is a useful one when ether cannot be employed, and it is inadvisable to use chloroform. It consists of alcohol, chloroform, and ether in the proportions by volume, of 1, 2, and 3 of sp. gr. ·838, 1·497, ·735 respectively. Martindale suggests a better mixture, consisting of the ingredients of the sp. gr. ·795, 1·497, ·720 respectively, the mixture having a sp. gr. of 1·01. The alcohol probably acts simply as a menstruum, and may be replaced by eau de Cologne. When evaporated from a closed inhaler (a practice which is not to be commended), it is impossible to be sure that the vapours are given off in the proportion in which the ingredients occur in the mixture, ether probably coming off first and chloroform being taken in increased strength as the administration proceeds. It is best, therefore, to use the mixture from a cone, a Rendle's mask, or some such simple contrivance, pouring a few drachms on at a time, or it may be used with a Skinner's mask, the anæsthetic being spilt on from a drop bottle. I have found Allis's open inhaler very useful for this mixture. The dangers of the A.C.E. mixture are those incident to chloroform, although they are lessened by the dilution of that ingredient in the mixture.

The A.C.E. mixture is taken easily by children, and is convenient for use in parturition. It should be given to the patient only in the supine position, and well diluted with air. No apparatus is necessary, but the employment of one of those mentioned above prevents waste, and enables the administrator to control the supply according to the requirements of the case. It is a useful plan, if nitrous oxide gas is not available, to commence with the A.C.E. mixture from a cone or Skinner's mask, and as soon as excitement appears continue and maintain the anæsthesia with ether from a Clover's or Ormsby's ether-inhaler. Any duskiness shows the dilution of the vapour is insufficient.

Deaths under this mixture have been imputed both to primary syncope and to neuroparesis from poisoning of the centres in the medulla oblongata.

Various other mixtures of ether and chloroform have been proposed and found favour in some cliniques. Their behaviour depends upon the proportion of chloroform present in them. Thus—

Vienna mixture, 6 parts of ether to 2 chloroform, or, in hot weather, 8 parts of ether to 1 of chloroform are used.

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[1] *Clinical Journal*, 11th January 1898, p. 200.

Richardson's mixture, 2 of chloroform, 2 of alcohol, and 3 of ether.

Dr. Schleich (Berlin) has introduced mixtures of sulphuric ether, petroleum ether, and chloroform in the following proportions:

	I. (Slight anæsthesia).	II. (Medium).	III. (Deep).
Sulphuric ether	180 parts.	150 parts.	80 parts.
Petroleum ether	15 „	15 „	15 „
Chloroform	45 „	45 „	30 „
Boiling-point	38° C.	40° C.	42° C.

His view is that by giving a mixture, the boiling-point of which is nearly the temperature of the body, its inspiration is easily achieved, and it rapidly leaves the body without producing any deleterious effects. The theoretical views have not, however, been altogether supported by clinical experience. Schleich used the mixture from an Esmarck's mask. No. I is suggested for light transient anæsthesia in the case of minor surgery, II. when a medium or somewhat deeper anæsthesia is deemed necessary, and III., when profound surgical anæsthesia is required. Schleich claims that no unpleasant complications or after-effects have attended the use of his mixtures, but a somewhat extended employment of them in some of the hospitals in the United States has not wholly borne out his contention.

Nitrous Oxide Gas.—This anæsthetic is valuable for short operations about the mouth, giving a period of anæsthesia of from twenty to forty seconds in duration, when, however, the face mask can be taken off the face, and reapplied from time to time, or when the nitrous oxide is mixed with air or oxygen a more prolonged period of anæsthesia may be obtained, limited indeed only by the amount of gas procurable. For very lengthy operations the cost of the material and the difficulty of conveying large numbers of the steel bottles in which the gas is kept renders its use inadvisable. Gas mixed with air or oxygen has been used for operations for the relief of empyema thoracis, for radical case of hernia, and many other major operations. There is more muscular rigidity than in the case of ether or chloroform, but it is alleged the after-effects are slight.

Gas may be given from a gasometer or from steel cylinders in which it is compressed into a liquid. Upon turning the screw tap closing the cylinder the pressure is removed, and the liquid at once becomes gasiform. The gas is made to travel along a tube into the Catling's bag, which has a capacity of two gallons, and is respired from this bag by the patient through an accurately fixing face-mask. It is possible to give nitrous oxide by the "open method" in cases in which patients are terrified by a closely applied mask. Flux's[1] plan is to tuck a towel below the chin and back to the ears and up to the level of the eyes, taking care that close apposition takes place all round the lower part of the face. Gas is then allowed to fall down from the delivery tube of the gas apparatus into the space between the towel and the face. As the gas is heavier than air it sinks, displacing the air, and the patient inspires it quietly. Flux has devised celluloid masks which replace the towel. The open method, although very successful in some cases, does not, in my opinion, ensure so perfect or deep an anæsthesia as when a mask fitting tightly on the face is employed. The signs of anæsthesia under gas are, slow regular respiration, stertorous breathing, and spasmodic twitching of the muscles. Slight dilatation of the pupils and fixation of the ocular globes occur in full anæsthesia, phenomena which are best seen when the gas is given combined with air or oxygen. It is, however, always wise to give air intermittently with nitrous oxide gas to avoid asphyxial complications. This can be done by admitting air when muscular jactitation commences by opening the air inlet valve or lifting the face-piece for one inspiration. When Hewitt's method of giving nitrous oxide with oxygen is employed a special apparatus devised by him is requisite. It consists of a bag divided into two, each half being connected by tubes respectively with the nitrous oxide or oxygen supply. The gases are allowed to mingle in a chamber in front of the bag in a proportion controlled by a movable index. The patient is made to respire nitrous oxide gas with a small quantity of oxygen, the amount being increased as the anæsthesia progresses. Excess of oxygen produces excitement, deficiency cyanosis. A slightly prolonged anæsthesia is obtained in this way, and complete avoidance of muscular jactitation, so that the actual period of operation is longer. Patients are a trifle more dazed, and in some cases complain of nausea, after the use of oxygen.

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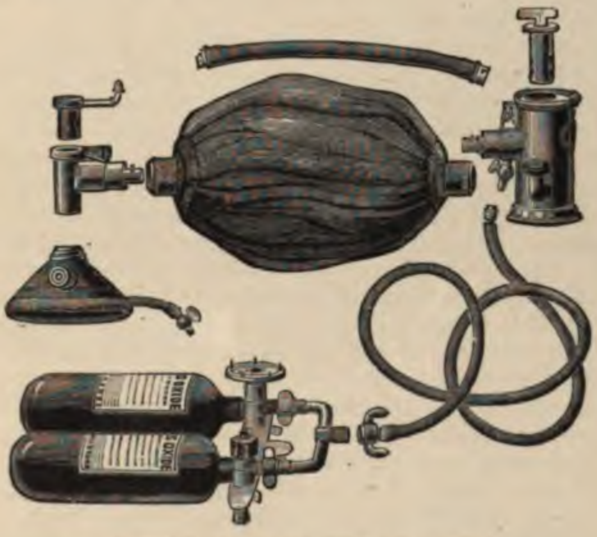
[1] *Lancet*, Feb. 4th, 1899.

The consideration whether diseased conditions in patients contra-indicate the use of nitrous oxide gas is of less importance than in the case of other anæsthetics, as the risks of this anæsthetic when properly given may be said to be nil. Practically any one can take gas, but where obstructive lung disease is present, or when there is pre-existing cyanosis or dyspnœa, it is better to employ oxygen and nitrous oxide combined. Muscular and alcoholic subjects are not always satisfactory patients when oxygen is used; anæmic people and women and children, on the other hand, are remarkably good subjects. It is a common error to suppose that pregnant women cannot take gas with impunity. Certainly up to six months of pregnancy no risk exists. After that it may be questioned whether or not it is wise to permit of any surgical proceeding, but if such must be adopted there is far less risk in using nitrous oxide gas, or other anæsthetic, than would arise if the pain of the operation were allowed to be felt.



Gas and Ether apparatus with its parts separated.

Nitrous Oxide before Ether.—The patient is put under gas until the breathing becomes regular and deep, usually after ten or twelve respirations, then the ether is turned on and gradually more and more ether given until full etherisation exists. The cyanosis which commonly shows itself at this stage is at once removed by lifting the inhaler from the patient's face and allowing him one or two inspirations of air.

The woodcut represents the apparatus which is commonly employed by the writer. It is depicted with its component parts separated as they should be after use and when about to be cleansed before being again used. The apparatus can be thus rendered absolutely aseptic.

It consists of two horizontal fifty-gallon cylinders of nitrous oxide gas, although one cylinder or even a smaller (twenty-five gallon) cylinder can be used, and the ether inhaler connected to the gas supply by a length of india-rubber tubing. The ether inhaler is composed of the metal chamber into which the tubular valve is pushed down, when in use, a central tube running through the india-rubber bag and connected at one end with the ether chamber and at the other with the metal mount which carries the face-piece. This central tube conveys the ether vapour and ensures adequate strength of vapour. The Catling's bag holds the gas or air, as the case may be, and permits of free inspiration. When the patient is once fully under the influence of the ether, the gas tube is disconnected and the tap left open, thus permitting air to enter. This prevents cyanosis and ensures comfort in breathing.

Prolongation of Nitrous Oxide Anæsthesia.—This may be done by using ether, as above, or by giving the gas intermittently, or when the mouth cannot be covered by forcing gas up through a bent tube which is held in the mouth (Coxon). A nose-cap (Coleman) allows the patient to go on inhaling the gas after the mouth-piece is removed; or the same end is effected by introducing a soft catheter through the nostril and causing gas to pass through it into the air-passages.

Complications of Nitrous Oxide Gas in Anæsthesia.—Syncopal attacks are extremely rare, although in feeble subjects they may recur, especially if the tongue is allowed to fall back. The horizontal posture and dragging forward the tongue soon restore animation. Interference with respiration from entrance of foreign bodies or falling back of the tongue is a more frequent complication.

It is important that all clothing be loosened and that the stomach is not distended with semi-digested food at the time of the administration. The position of the patient, more especially as regards his head and neck, should be so adjusted that no impediment is imposed to respiration. To ensure this in the sitting posture the body should be slightly extended on the pelvis, the head slightly extended on the trunk and maintained in the middle line. Under no circumstances should loose bodies, *e.g.* teeth, be left free in the mouth. Gags should always be secured out of the mouth and be placed upon teeth sufficiently strong to withstand spasmodic contraction of the jaw muscles. In the case of epileptic patients a fit is liable to occur as the patient is passing into narcosis, and unless the gag is properly secured, carefully placed, and sufficiently strong it may be bitten through or may displace teeth.

Bromide of Ethyl, although much in vogue at one time, has been shown to be dangerous, and it can only be employed for brief operations. Its action appears to be cardio-depressant, and to lessen arterial tension (Wood and Thornton), so it is not to be recommended for routine use.

It is an unstable body, rapidly undergoing decomposition if exposed to light and air; when at all coloured it should not be used, as impure specimens contain free bromine, and are both irritating and dangerous when respired. Patients pass very rapidly under the influence of bromide of ethyl with little excitement, but muscular rigidity usually persists, although some authorities deny this. The ocular reflex is not lost, and owing to the great volatility of bromethyl the anæsthesia is very brief, disappearing almost as soon as the inhalation is closed. It should be given from a closed inhaler, all air being excluded. Silk, following the plan of Schneider, pours one to one and a half drachms upon the sponge of an Ormsby's inhaler, and only removes the face-piece when palatine stertor is feared. The patient should abstain from food, and be in the horizontal position. It must not be used for other than brief operations. Bromethyl has been extensively used in the operation for removal of adenoid post-nasal growths. Although several specialists have so used it, and placed their patients in the vertical position, the undoubted tendency to syncope which this anæsthetic produces, and the increased hæmorrhage which its use involves, do not recommend it for this class of cases.

Pental, possessing the same formula as amylene, a body introduced by the late Dr. Snow as an anæsthetic, has been carefully investigated by Cerna, who regarded it as a dangerous anæsthetic, from its cardio-depressant properties. It is said to cause albuminuria and even hæmaturia. Others deny these allegations, and regard pental as a useful anæsthetic for children. I cannot think it possesses any advantages over chloroform; its smell is disagreeable, and its after-effects are no less than those following chloroform given for the brief operations in which pental has been employed. It is given in the same way as chloroform, but a more concentrated vapour is used.

Local Anæsthesia. — Extensive experimentation has led Schleich to propose a plan whereby not only the superficial but the deeper structures can be rendered anæsthetic by his "infiltration method." His suggestion and that of Reclus have been modified, extended, and improved upon.[1]

The method consists in a careful sterilisation of the skin, the employment of sterilised solution of cocaine in saline solution with morphine added.

Schleich's solutions are—

	I.	II.	III.
Cocain. Hydrochlor. .	0·2	0·1	0·01
Morph. Hydrochlor. .	0·02	0·02	0·005
Sod. Chlor. . . .	0·2	0·2	0·2
Aq. destill. ad . .	100·0 (sterilisat.)	100·0	100·0

Eucaine B is recommended by Braun and Heintze, with omission of the morphine, this anæsthetic being selected as it can be boiled without decomposition, and as it appears to be less toxic than either cocaine or eucaine.

A solution containing Eucaine B, Sodium Chloride, and warm water is, after being boiled, kept warm in a glass vessel, a large, specially-constructed hypodermic syringe holding 5 c.c. is then filled, the needle is carefully inserted endermically for its length, and then gradually withdrawn, the fluid being forced into the skin until a wheal results. The needle is reintroduced into the periphery of this wheal, and a further wheal made along the line of the proposed incision. As now the skin is anæsthetic the needle is introduced in the same line subdermically, further injection being made, and afterwards the deeper tissues are infiltrated in the same way. An anæsthetic œdema is thus produced, which in a few minutes makes cutting painless.

When structures are being handled which cannot be injected, such as the peritoneum, the solution should be injected over them from time to time from the syringe. The anæsthesia lasts from ten to twenty minutes, but, of course, can be renewed by fresh injections. It is of the utmost importance to ensure asepsis in the solution and in the apparatus employed. Loose tissues such as those of the tongue, etc., should never be infiltrated; fatal œdema of the glottis has resulted from inattention to this rule. Patients subjected to operation under the infiltration method occasionally become extremely faint and may vomit. Save for this, there is no reason why they should not have their ordinary meals, indeed it is recommended by some German surgeons to give a full meal and alcohol before the infiltration takes place. When the hollow viscera are operated upon, this method does not at present seem to be always successful. It answers best where the area of operation is small, or where the tissues are sufficiently dense to avoid the œdema rapidly forcing the infiltrated fluid into adjoining areas; it is not successful for inflamed tissues, and there is danger when it is employed in the region of new growths lest the *materies morbi* be injected into the lymph spaces around the region operated upon. Most of the major and minor operations of surgery have now been performed under the infiltration method of local anæsthesia. The shock to the patient and the strain upon the nervous system from the ordeal of conscious, even if painless, presence at the operation on their own body constitute the chief drawbacks to the method. Undoubtedly in cases in which general anæsthetic is for any reason considered undesirable, the infiltration method offers a valuable alternative course.[1]

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[1] Full details of this method can be found in *Schmerzlose Operationen*, von Dr. C. L. Schleich. Berlin, 1899.

[2] The solution of Eucaine B is 1 part in 1000 of normal saline solution (sodium chloride eight parts), by weight.

The injection of cocaine, even in small doses, is never free from risk. Fatal syncope has followed from quite minute doses. There is also the further danger of inducing the cocaine habit. There are no signs by which a patient can be discriminated as one for whom cocaine should not be used. In cases of cocaine poisoning after injection, artificial respiration and the employment of cardiac stimulants should be resorted to (*see* "Cocaine").

Local anæsthesia by freezing the tissues was suggested by the late Sir Benjamin Ward Richardson, who employed a special form of ether and sprayed it over the part to be made insensitive. Ethyl chloride and methyl chloride, or in combination, have been used in like manner. These bodies are kept in glass tubes, and the liquid allowed to flow over the skin, which is thus rendered insensitive by the cooling caused by the rapid evaporation of the anæsthetic. This means of securing painlessness is not devoid of drawbacks, and is only serviceable in the most trivial of operations.

Analgesics and Anodynes.—*Analgesic remedies* diminish the sensibility to pain throughout the body generally.

Anodynes may act in this way and so include the analgesic group, the general anodynes; or relieve pain locally by action upon the terminations of sensory nerves, the local anodynes or anæsthetics. The latter group alone is frequently described nowadays by the term "anodyne."

Actions.—The difference between a general anæsthetic and an analgesic lies in the fact that an anæsthetic not only removes the sensation of present pain, but also abolishes sensibility to subsequent painful impressions, while an analgesic diminishes the acuity of the nervous sensory mechanism for the feeling of pain, especially if it be due to central causes, or to irritation of the sensory communications between the brain and the skin, but is not able to abolish the sense of pain produced by immediate injury or pressure. Local anodynes similarly soothe painful impressions already existent; local anæsthetics absolutely negative the power of feeling pain, whatever violence be done to the area affected by them.

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[1] For an account of Braun and Heintze's methods see "A Clinical Lecture on Local Infiltration Analgesia," by A. E. Barker, F.R.C.S., *Lancet*, Feb. 1899.

Many of the analgesics and anodynes, if given in large doses, affect the higher cerebral centres; the majority of the synthetic analgesics act also upon the thermo-taxic mechanism and diminish thermo-genesis (antipyretics).

Pain is primarily caused by irritation of the peripheral sensitive cells of the sensory nerve-filaments, or of the filaments in nerve-trunks themselves; while, possibly, injury to or pressure upon the sensory filaments as they pass up the spinal cord can register painful impressions in the brain.

It is uncertain whether the nerves of sensation can be painfully affected during their passage through the lower brain on their way to the cerebrum, probably they are receptive in the basal portions at least, while the occurrence of subjective symptoms of pain in many cases, which pressure upon the nerve filaments in the dura or the pia mater appears to be insufficient to account for, might suggest the possession of local sensibility.

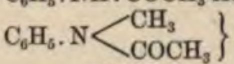
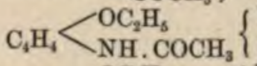
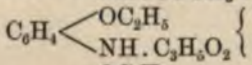
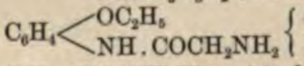
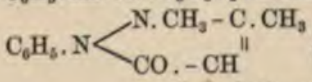
The majority of the synthetic analgesic drugs act upon the pain-conducting paths in the spinal cord, but at the same time affect both the brain and the blood. The gray matter of the cerebrum is slightly depressed and the perception of impressions lessened. The great medullary centres are depressed, thus leading sometimes to respiratory and cardiac disturbances, or to collapse from a more or less rapid dilatation of the peripheral blood-vessels, owing to an action upon the vaso-motor centre. Hæmoglobin is altered by many of them, causing disintegration of the red blood corpuscles. Above all, drugs of this class are peculiarly apt to cause different effects upon different individuals, or upon the same person on different occasions.

Opium, its alkaloids, and bromides act by lowering the excitability of the sensory nerves as well as of the cord and brain. Belladonna and hyoscyamus act on the terminal sensory organs.

TABLE OF ANALGESICS AND ANODYNES (after Stockman).

(A) *Of General Action.*

Synthetic Analgesics.

Source.	Derivative.
1. Phenol . .	$C_6H_5.OH$.
	$C_6H_5.COOH$ Benzoic Acid.
	$C_6H_4.OH.COOH$ Salicylic Acid.
2. Anilin . .	$C_6H_5.NH_2$ Anilin.
	$C_6H_5.NH.COCH_3$ Acetanilid, Antifebrin.
	 $C_6H_5.N\left<\begin{matrix}CH_3\\COCH_3\end{matrix}\right.$ Methylacetanilid, Exalgin.
	 $C_4H_4\left<\begin{matrix}OC_2H_5\\NH.COCH_3\end{matrix}\right.$ Ethoxyacetanilid, Acetphenetidin. Phenacetin.
	 $C_6H_4\left<\begin{matrix}OC_2H_5\\NH.C_3H_5O_2\end{matrix}\right.$ Lactyl Phenetidin. Lactophenin.
	 $C_6H_4\left<\begin{matrix}OC_2H_5\\NH.COCH_2NH_2\end{matrix}\right.$ Amido-phenacetin. Phenocoll.
3. Phenyl-hydrazin	$C_6H_5.HN.NH_2$ Phenyl-hydrazin.
	$C_6H_5.HN.NH.COCH_3$ Pyrodin. Acetyl-phenyl-hydrazin.
	$C_6H_5.HN.N.C_5H_8O_2$ Antithermin.
4. Phenazonum .	 $C_6H_5.N\left<\begin{matrix}N.CH_3-C.CH_3\\CO.-CH\end{matrix}\right.$ Phenyl-dimethyl-isopyrazolon. Antipyrin, Phenazonum.
	Salipyrin, Salicylate of Antipyrin. Tolypyrin, Toly-dimethyl-pyrazolon. Tolysal, Tolipyrin Salicylate.

Opium and its alkaloids; Bromides; Belladonna and its alkaloid Atropina; and the various kindred alkaloids of Hyoscyamus, Stramonium, etc.; Gelsemina; Chloroformum; Chloral hydras.

(B) *Of Local Action.*—Morphina, Codeina, Cocaina, and Aconitina.

If the table shown be consulted, it may be seen that the more common analgesic drugs are derived from phenol and its compounds, or the products made from it. From phenol, anilin can be prepared; from anilin, phenyl-hydrazin; from the latter, phenazonum. Of these, anilin and phenyl-hydrazin are very poisonous, and their derivatives are apparently more dangerous than the compounds made from the other chemical kernels.

Uses.—There is a great divergence of opinion as to the member of the synthetic group of analgesics which acts most satisfactorily, and at the same time whose administration is attended by the least risk of unpleasant symptoms. Some place phenazonum first, some phenacetinum; others again pin their faith to acetanilidum, lactophenin, salophen, exalgin, etc. They all have an advantage in that they do not disturb the mental processes, although affecting the lower cerebral ganglia.

Phenazonum and phenacetinum, when prescribed along with caffeina or caffeinæ citras, may be regarded as those which can be most frequently given without unfavourable sequelæ following their administration. The derivatives of interaction between salicylic acid and phenazonum have been used with success in cases of rheumatism and in those of rheumatic habit.

Opium and its alkaloids, morphina and codeina, are used, generally or locally, to relieve actual pain, whatever be the cause. Bromides act more by lessening perception than by annulling the sense of pain. Two drugs appear to have a special influence upon the fifth cranial nerve in relieving painful sensations referred to it, *e.g.* butyl-chloral and gelsemium.

Anaphrodisiacs.—These are remedies which diminish sexual desire or power. The principal anaphrodisiacs are—the bromides, especially bromide of potassium, iodide of potassium, camphor in the later stages of its action, alkalies in large doses, administered for a continued period, and, generally, all measures which depress the "tone" of the system, such as starvation, purgatives, bleeding, wasting diseases, excessive mental or bodily labour.

These may act directly on the genital organs, or upon the genital centres of the brain and cord, or indirectly through the circulation. Anaphrodisiacs are indicated in cases where sexual desire is excessive, or when legitimate means of gratification are absent. However, in diseases characterised by a morbid exaggeration of sexual desire, combined with loss of self-control (inhibition), moral and hygienic treatment is of the greatest importance, and anaphrodisiac drugs are useful as adjuncts only.

Apart from disease, advantage may be taken of the well-known physiological fact that severe muscular exercise (as in "training" for sports) or mental labour (as in studying for examinations) are attended by temporary diminution or absence of sexual desire, in cases where it is not feasible to carry out St. Paul's advice that it is better to marry than to burn.

Such depressant measures as the administration of very large doses of alkalies are not made use of in practice for controlling morbid sexual appetite.

Aneurysm.

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Anatomy of Aneurysm.—Aneurysms occur of all sizes, from that which is just visible (pulmonary, cerebral) up to that even of an adult head.

Number.—Whilst miliary aneurysms are often numerous, large aneurysms are usually single. It is not rare, however, to find (1) symmetrical arteries (especially the two popliteals) dilated, (2) two to four aneurysms upon the same main trunk, *e.g.* femoro-popliteal, or (3) an external aneurysm combined with an internal. Three and even four distinct sacs have been found springing from the aortic arch—itself uniformly dilated. It is said that as many as sixty-three aneurysms have been counted in one subject. Cases such as these have caused some to use the expression "aneurysmal diathesis," a condition which, like that in which multiple fractures occur, has as yet no anatomical basis.

Form.—An aneurysm may be "fusiform" when the vessel is uniformly dilated, or "saccular" when only a limited part of the wall is affected.

Sometimes it is of a "mixed" type, due to the development of a saccule from a fusiform aneurysm.

Structure of the Sac.—In both forms of aneurysm, but more especially in the saccular, the sac is formed, not by any part of the original vessel wall, but by a more or less dense layer of fibrous tissue which has been derived from the following sources:—

(1) From inflammatory and reparative processes arising in the original arterial tissue and tissue of the sac. (2) From organisation of clot adherent to its inner surface. (3) From the connective tissue of parts with which the aneurysm has blended. In this tissue nerves and other important structures may be involved.

The situation of an aneurysm, the nature of the surrounding tissues, and the rapidity of its growth, are the main factors determining the relative thickness of its wall.

With regard to the artery from which an aneurysm has sprung, it was until recently supposed that it was always sclerotic, degenerate, and unsuitable for ligature; but examination has shown that theory to be wrong. The vessel may be diseased, it is true, but it is just as probable, if not more probable, that the popliteal in a case of popliteal aneurysm will be fit for ligature as that the femoral in Scarpa's space will be so.

Contents.—Fluid blood and laminated clot in varying proportions constitute the contents of all aneurysms. Organised connective tissue is met with very exceptionally, owing to the diminished vitality and deficient vascularity and reparative power in the wall of the vessel. The relative proportions of laminated clot and fluid blood vary according to the form of aneurysm, the laminated clot being naturally less pronounced in fusiform cases owing to the free circulation through the swelling.

Etiology.—There are two factors in the production of an aneurysm:—(*a*) The blood-pressure; (*b*) the resistance of the arterial wall.

(*a*) *Heightened Blood-pressure.*—Normal blood-pressure may rupture elements in or even the whole thickness of a weakened artery; heightened blood-pressure will do this the more easily, and is itself a cause of arterial disease. Eppinger goes so far as to say that all spontaneous aneurysms are traumatic, in that they begin in slight ruptures of the media.

Heightened blood-pressure is produced for longer or shorter periods by violent efforts or prolonged exertion. In many of these not only is the heart acting more rapidly and strongly, but it is opposed by increased resistance, owing to the steadily maintained contraction of large numbers of muscles compressing the intra-muscular capillaries. Efforts out of proportion to the strength of the individual, undertaken without suitable "training," especially by those who are approaching middle age, may easily result in slight ruptures of the media—lesions which probably cause no immediate symptoms, and which may perhaps heal and remain sound in many cases. Those who earn their living by a series of frequently repeated strong muscular efforts maintain a more or less constantly increased blood-pressure which sooner or later tells upon their vessels and renders them, as a class, unduly liable to aneurysm. Permanent arterial strain results from plethora, hypertrophy of the left ventricle, and from the general angio-sclerosis of Bright's disease, with which alcoholism, plumbism, and the gouty diathesis must be associated etiologically. Effort will, of course, still further raise the blood-pressure in these cases.

Indeed, it is not difficult to believe that more or less serious ruptures of arterial tissues occur; the difficulty is rather to understand why they do not happen oftener.

(*b*) *Impaired strength and elasticity of the arterial wall* may be the result of—

1. *Congenital Defect.*—In some rare cases of aneurysm in healthy children and young people, no known cause of arterial weakness has been recognised. It is possible that the media may have been congenitally weak. Eppinger states that he has demonstrated relative weakness of the media in certain of the healthy arteries he examined.

2. *Injury.*—It is said that non-penetrating incised wounds of vessels may allow the inner tissues to protrude and form an aneurysm ("hernial"). Small penetrating wounds may heal and the scar yield later. The effects of distending strain have been considered. Longitudinal strain—as when the knee is over-extended till the posterior ligament cracks—will rupture the inner coats of the popliteal. The greater frequency of aneurysm on the right side of the root of the neck seems due to strain on the great vessels from the more frequent and stronger use of the right arm; and it is probable that the great frequency of popliteal among external aneurysms is connected with the frequent and strong extension movements of the joint—the artery being fixed by its articular branches. Sometimes a strong extension move-

ment, *e.g.* jumping a ditch, seems to rupture something, and a popliteal aneurysm follows. Squeezes and contusions may rupture more or less of the inner tunics: occasionally the pulse disappears from large vessels after a contusion. Even digital and instrumental compression have been followed by aneurysm. So, too, has the crushing action of an aseptic ligature, which has softened or come undone before occlusion of the vessel has occurred. Vascular weakness renders injury and dilatation from these causes more likely.

3. *Arteritis by infection from without, usually coupled with loss of natural support.* The commonest examples of this are the small aneurysms of the pulmonary artery in the walls of a tubercular cavity. Tubercular inflammation rarely attacks large vessels.

Septic ligature with rupture of the inner coats was often followed by an acute arteritis with great swelling and softening of the wall which usually ended in secondary hæmorrhage, but sometimes in dilatation. A similar acute dilatation may affect uninjured arteries in cases of spreading suppuration. Thus an abscess formed above the wrist after erysipelas; it burst, and free bleeding followed from an aneurysm the size of a walnut on the radial. Small "erosion" aneurysms have been met with in the floor of gastric and other ulcers; but large vessels (*e.g.* femoral in sloughing bubo) when eroded usually show little dilatation before they burst and bleed.

4. *Arteritis by Infection from within.*—The above causes account for very few aneurysms; that which is about to be considered accounts for a larger number, though still not for many—viz. for most aneurysms occurring in children and young adults, together with some in more advanced life. When an infective embolus lodges in a vessel it induces an acute arteritis similar to the septic form; which, also, may result in bursting (and formation of an arterial hæmatoma) or in dilatation. The embolus irritates mechanically, especially if it contain calcareous masses, and chemically by toxins with which it may be soaked; and thus prepares the way for invasion of the wall by the cocci in the embolus—the important irritant. Ponfick, however, attributed certain aneurysms examined by him to ulceration of the vessel from the mechanical irritation alone of calcareous masses.

Aneurysms from embolism usually occur in the young, and on arteries of medium size; they are often multiple; there is almost always disease of a left valve, and usually the patient presents the symptoms of endocarditis maligna. But this is not always the case, for the same cocci may be found in warty as in ulcerative endocarditis. The impact of the embolus is often accompanied by sharp pain, and circulatory disturbance is common.

5. *Chronic Arterial Disease.*—Endarteritis deformans, Atheroma, Arterio-sclerosis. (*See* "Vessels.")—This chronic inflammatory and degenerative process of the intima plays an important part in the development of aneurysm.

Until recently the arterial changes which have received these names were regarded as primarily inflammatory, the chief irritant being the strain of the blood-pressure, especially when heightened. The inflammatory products effused in the subendothelial layers were poorly nourished, and tending strongly to fatty degeneration were the most *striking lesions.* Occasional thickening of the adventitia were recognised, but little notice was taken of changes in the media; loss of elasticity, general dilatation and lengthening of the vessels affected was well known. No doubt was felt but that this condition, which is present in the great majority of aneurysms, was the passive cause of the dilatation—uniform loss of strength and of elasticity leading to fusiform aneurysms, local weakening by bursting of an atheromatous focus or shifting of a calcareous plate resulting in a saccular pouch.

Against this view it was pointed out that aneurysm is a disease of early middle life, atheroma of late middle and old age; that atheroma is very common in all countries, but aneurysm is not; that atheroma is equally common in women and in men, but aneurysm is far more frequent in men; that aneurysms occur in which there is no atheroma or none in association with the aneurysm. Good answers were not difficult to make. Atheroma is not very rare under thirty, and is commonly found in cases of aneurysm in young adults. It is difficult upon any theory to account for the varying incidence of aneurysm in different countries. Atheroma is equally common in the two sexes only after middle life; before this, far commoner in men. Lastly, it was not asserted that atheroma was the only cause of aneurysm.

Many years ago v. Recklinghausen advanced the view that the essential lesion in aneurysm was rupture of the media. His pupils, Eppinger and Manchot, have done much to establish it by the frequency with which they have demonstrated ruptures of the elastic lamellæ in early aneurysms. They regard the presence of atheroma in cases of aneurysm as fortuitous. What is essential to the production of an aneurysm is either congenital or acquired weakness of the media, so that it will rupture under some strain to which it may be exposed.

Thoma has made an exhaustive study of arterio-sclerosis. Its histology is very complex, and consists of fatty degeneration of connective tissue and muscle cells and granular change of elastic fibres; occasionally ruptured elastic fibres; frequent localised or diffuse inflammatory foci, the result of injuries and strains; and lastly, fibroblasts and young connective tissue, indicating efforts at repair. Thoma concludes that the most striking lesions of endarteritis deformans are to be regarded as compensatory for less obvious, weakening lesions, chiefly of the media. Aneurysm, according to this view, occurs only when the compensation fails. Among other proofs of the compensatory nature of arterio-sclerosis Thoma showed that gelatine casts of atheromatous vessels, made under normal blood-pressure, have smooth surfaces; they should have lumps opposite the plaques if these were weak spots.

The very frequent association of aneurysm and arterio-sclerosis does not necessarily imply that the latter is the cause of the former: they may both be the result of the same cause, viz. weakness of the media, and arterio-sclerosis may really be preventive of aneurysm. If atheromatous plaques degenerate and burst, or calcify and are swept away, the compensation will, of course, be reduced considerably and a slighter strain will cause dilatation.

Syphilis is a cause of chronic endarteritis: is it a cause of aneurysm? It is best seen in the circle of Willis and its branches, where it produces an endarteritis and tends to cause occlusion by irregular gummatous thickening. But it certainly may affect the aorta and great vessels rising from it, and gummata may produce changes, such as stretching and rupture of the media, from which aneurysms arise. There can be no doubt that some aneurysms owe their origin to syphilis, but there is great doubt as to how many do, and as to whether the disease affects all vessels or only certain ones.

Geographical Distribution.—This is by no means uniform. External aneurysms are said to be very rare in Germany and Belgium, more common in Italy and France, in which latter country it is said to be increasing, most frequent in Great Britain and Ireland and in America.

Situation.—Aneurysms of the aorta seem to be three to four times as frequent as all "external" aneurysms taken together. The thoracic aorta, especially the arch, suffers most frequently, because the pressure here is highest; chronic and acute arteritis are most common and severe; and it is liable to lateral strain through the great vessels arising from it, especially on the right. Of "external" aneurysms the popliteal is much the most common. Of 142 such aneurysms collected from recent reports of four London hospitals, 79—well over half—were popliteal, 20 femoral (15 in Hunter's canal), 7 ilio-femoral, 8 axillary, 6 subclavian, 6 common carotid. The striking excess of lower limb aneurysms over those of the upper limb may be connected with the higher pressure and less perfect nutrition which prevail in the former.

Sex.—Of these 142 cases 11 were in females = 1 in 13.

Age.—The age is given in 72 popliteal cases. There were 2 at twenty-five or under, 14 at thirty, 20 at thirty-five, making exactly half (36) by thirty-five; the next five-yearly periods showed 11, 10, and 10; there were 3 before fifty-five, 1 before sixty-five, and 1 at seventy. The aneurysm age may be said to run from thirty to fifty, *i.e.* during the period before muscular strength fails, and when therefore the hardest work and exercise are persisted in, but during which also vascular disease and degeneration begin and often reach a high development in those who are predisposed. It is true that atheroma is most common and most severe after fifty, but by this time loss of strength and energy necessitates the abandonment of the most laborious occupations.

Occupation.—Aneurysms are most common among those whose occupations require the most powerful efforts. Statements connecting special aneurysms with certain occupations—*e.g.* popliteal with postillions—are apparently mere impressions.

The Signs and Symptoms of Aneurysm.—The patients' attention is not uncommonly attracted by a sense of something snapping or giving way during an effort. More often the first symptom is pain in the part, or pain, weight, or numbness in the area of distribution of some nerve pressed upon, or a sense of weakness in a part. In other cases an unusual beating or an obvious pulsating tumour is the first thing noticed; much less commonly œdema of a distal part, as the foot.

The signs of aneurysm divide themselves naturally into (1) those due to the presence of a tumour-like swelling, and (2) those which indicate the communication of this swelling with an artery.

1. The tumour-like swelling in external aneurysms varies usually from the size of a marble to that of a fœtal head. It is rounded, oval, or fusiform in outline; hour-glass-shaped owing to constriction by Poupart's ligament or the clavicle; lobulated from large secondary saccules. When the mass can be well grasped, it is found to be distinctly outlined, firm, tense, elastic, and pulsating; but its firmness varies much with the thickness of the sac and lining clot, and its tension with the blood-pressure. In the diagnosis between saccular and fusiform aneurysms, palpation is apt to be very misleading.

Pressure effects.—In the process of development of an aneurysm, all the structures that can be displaced or stretched are thus affected, and when this is no longer possible, atrophy, erosion, and absorption of the compressed tissues follow. These pressure effects will naturally vary with the position of the swelling, and they are important as usually constituting the basis of the subjective symptoms complained of.

Typical pressure symptoms are specially well seen in cases of aortic aneurysm, to which article the reader is referred. In any given case the symptoms of pressure on nerves, vessels, and other tissues will be readily intelligible on consideration of the anatomy of the part.

2. Signs due to communication with an artery.

Situation.—The swelling is in the line of a main artery and cannot be so displaced from the vessel as to cause the disappearance of the signs to be noticed next.

Expansile pulsation is the most characteristic sign. It is due to the alternating systolic distension and diastolic contraction of the sac. It is easy to detect when the swelling can be grasped or a finger be laid upon opposite poles; but when fingers can be placed only close together upon a small area of the surface, it may be impossible to be sure that pulsation is expansile, and not due to the driving of some mass, lying on the artery, outwards between the fingers. In large sacs pulsation may be more marked at the softer, more prominent parts.

A systolic thrill is not uncommonly felt, marked or faint, over the whole surface or only at one spot.

A diastolic shock is often felt in aneurysms at the root of the neck—like a thud *felt*. It nearly coincides with the second sound. *Accentuation of the heart-sounds*, especially the second, is frequent over the areas of these aneurysms.

A systolic bruit can generally be heard in external aneurysms, varying greatly in quality and intensity; rarely, a *diastolic bruit* also occurs (to-and-fro murmur). In fusiform aneurysms the bruit is often specially loud and harsh. A bruit, being due to the passage of blood through the mouth of the sac, is most likely to be present, loud, and high-pitched when the aperture is small. It is heard equally all over the swelling, and moderate pressure does not increase it—as it would were the bruit due to compression of an artery beneath some superjacent mass.

Effects of Compression.—Proximal compression of the artery stops pulsation, and the aneurysm shrinks and becomes softer; it may be rendered still smaller by direct pressure—a method of examination which may bring about embolism, and which should not be practised on the carotid, at all events. On releasing the artery, the sac regains its full size—not suddenly, but in two or three beats—often with a thrill. The softening and shrinking of the sac may be taken as a measure of the rigidity of the wall, which is generally proportionate to the contained clot. Distal pressure increases the pulsation and tension of the sac.

No examination of an aneurysm is complete without a thorough examination of the heart and great vessels, of the superficial arteries, and of the urine. It is most important to eliminate as far as may be an internal aneurysm, and to discover any other external aneurysm which may exist.

PROGRESS AND NATURAL TERMINATIONS.—As a rule external aneurysms go pretty steadily on to one or other of their natural endings, usually rupture, rarely spontaneous cure. Halts and unexpected improvements may alternate with advance.

1. *Rupture.*—In the limbs usually and in the trunk sometimes, aneurysms tend chiefly toward the skin surface, the skin becoming thinned, red, or livid, and gradually ulcerating, when death may occur from hæmorrhage. Rupture of an aneurysm may also occur into the surrounding areolar tissue or into a joint, and, in the case of internal aneurysms, into a serous cavity, into a hollow organ, or on to a free mucous surface.

The symptoms of ruptured aneurysm are sudden severe pain and collapse—partly from the pain, chiefly from internal or external hæmorrhage. But the size of the aperture, the suddenness of its formation, the quantity of blood lost, and the rate at which it escapes, all vary, and the symptoms vary in proportion. For the slighter cases, the term "leaking" aneurysm is convenient, whilst "ruptured" is used for the more severe.

The physical signs of leaking or rupture into a cavity are those of slow or rapid accumulation of fluid in it; whilst bleeding into a canal may be revealed by escape of blood from an orifice. When the leaking or rupture occurs into the substance of a limb, or other solid part, the physical signs vary in degree but not in kind. They are most severe (as also are the symptoms) when a considerable free opening forms suddenly between the sac and lax unmatted connective tissue around. Blood accumulates in the tissues round the aneurysm and forces its way along the areolar planes till stopped by the resistance of the tissues, aided by clothing; the cavity round the sac enlarges till similarly limited. Except that the blood has escaped from an aneurysm, there is no difference between this state of matters and the hæmatoma which follows upon ruptures of arteries. The limb swells greatly and diffusely, chiefly in the neighbourhood of the sac; it is tense, does not pit, and feels heavy and dead. The sac is no longer to be felt, there is no pulsation, thrill, or bruit. Arterial supply and venous return are greatly impeded beyond the hæmatoma; there is no pulse in the arteries, the part is cold and pale, marbled by full veins, perhaps œdematous. Shortly, the physical signs are:—sudden and rapid swelling about the aneurysm with loss of its outline and pulsation, and obstruction to the circulation.

Moist gangrene of the part may ensue upon a diffuse hæmatoma; but it is surprising what serious wounds of large arteries, coupled with hæmatomata, stop short of this. After a period of great doubt and anxiety, the limb slowly warms and evidences of circulation reappear in the digits; the swelling is gradually absorbed, becoming more and more limited to the neighbourhood of the aneurysm, and as the hæmatoma becomes circumscribed, pulsation, bruit, and perhaps thrill reappear—not in the original sac, but in the connective tissue capsule, which has formed around the extravasation. Sooner or later, bruising may appear.

In the case of less diffuse hæmatomata there may never be any anxiety as to the life of the part, and the hæmatoma soon becomes *circumscribed*. The rupture may be evidenced only by more or less pain, enlargement and blurring of the aneurysm, with less obvious pulsation. Or the leaking may be slow and continuous and its signs slowly induced. Extravasation of blood is often accompanied by fever, a symptom very likely to mislead in doubtful cases. In a case of aneurysm in Hunter's canal which ruptured after treatment with Esmarch's bandage, the patient died with symptoms of septicæmia (*Univ. Coll. Hosp. Rep.* 1878).

Untreated, a circumscribed arterial hæmatoma tends to increase and rupture. It may suppurate.

2. *Spontaneous Cure.*—The natural cure of aneurysm consists in the formation of sufficient clot to fill the sac and the artery. As would be expected, this spontaneous cure is much more common in cases of saccular aneurysm than in fusiform, owing to the free circulation and associated absence of rest in the sac of the latter. Occasionally a complete cure is thus effected. In very rare cases a spontaneous cure may be directly induced by the accidental blocking of the mouth of the sac or of the artery by an embolus of clot detached from the sac. A similar effect is thought by some to have been produced by pressure of the sac on the artery above or below the sac-mouth; but many doubt that this occurs, and refer the result rather to spread of thrombosis into the vessels. The development of one aneurysm above another may so depress the circulation as to lead to the cure of the lower aneurysm. A limited extravasation about the neck of a sac is said to have brought about a cure. The above act by inducing stagnation in the sac; but inflammation round about a sac may cause arteritis and thrombosis of the artery whence it springs. Lastly, spontaneous cure may be effected by sloughing away of the whole aneurysm.

The signs of spontaneous cure are:—More or less rapid hardening of the whole sac, the outline remaining definite; loss of pulsation and bruit; in some cases—especially sudden—great circulatory difficulties, and even dry gangrene may ensue; the pulse beyond is lost. The sac should shrink steadily; it may cause great pain by pressure or traction on a nerve.

Complications during the Course of an Aneurysm.—1. *Cellulitis and suppuration* about a sac are rare. No instance occurred among 142 cases from the consecutive reports of four hospitals, going back twenty-seven and twenty-six years in the case of two. It is said that infection is predisposed to by rapid increase amid loose connective tissue, and by sudden coagulation of the contents of the sac; it seems more likely that swelling and clotting result from commencing inflammation.

Usually infection has been connected with the application of a ligature in the neighbourhood, with injection, electrolysis, etc.; sometimes it has followed rough handling or other injury; sometimes there has been no discoverable cause other than that the sac and its surroundings necessarily form a locus minoris resistentiæ. (For treatment, see p. 223.)

Gangrene of distal parts (moist and dry) may result (1) from pressure of a rapidly enlarging sac, usually becoming diffused upon veins and, to a less extent, upon arteries; (2) upon rapid coagulation blocking not only the sac and its artery but important collateral branches; (3) by embolism of the main trunk or its branches beyond the aneurysm. The signs need no enumeration here.

DIAGNOSIS.—As above shown, aneurysms may be met with in four conditions—(1) Typical, with fluid contents and pulsation well marked; (2) solid or almost so; (3) diffused or leaking; and (4) inflamed or suppurating. All have to be borne in mind, and the difficulties vary in each case.

In its TYPICAL STATE, aneurysm is simulated by *fluid and soft solid swellings*, especially cysts of all kinds and chronic abscesses, lying in such relation to main vessels as to receive a communicated pulsation. *All pulsating tumours are not aneurysms.* Every endeavour should be made so to move a suspected tumour from the artery as to show its independence. Pulsating cysts are generally lax, thin-walled, of indefinite outline; if they lap round an artery they may pulsate expansilely, but the pulsation is feeble as compared with that of a thin-walled aneurysm. The tenser a cyst becomes, the more does its pulsation assume the simple heaving character—a mere rising and falling—which alone solid tumours acquire. Such tumours may not always have pulsated. They have, as a rule, no bruit; but pressure with a stethoscope may, by driving the mass back on to the artery, produce a rasping systolic sound, removed on lightening the pressure or on applying it to some other point or in some other direction. Proximal compression of the artery stops all pulsation; but the cyst or tumour cannot be reduced by pressure, except in the case of cysts behind the knee, the fluid of which can sometimes be pressed into the joint—where it may be detected. There is no increase and no thrill on removing pressure from the artery.

Among SOLID TUMOURS simulating aneurysm *Exophthalmic goitre* is important. The enlargement of the thyroid in Graves' disease is not as a rule great; but it is often asymmetrical, the right side being the larger. It pulsates *proprio motu*, often has a marked thrill and murmur. The shape of the swelling, particularly the way in which it runs down and in to the thyroid isthmus, is suggestive of its nature; the isthmus, if not the opposite lobe, is almost always palpable; the pulsation is not so forcible as that of an aneurysm; but the great point is that the swelling rises and falls with the larynx in swallowing. The presence of exophthalmos and other signs of Graves' disease should be looked for.

But the tumour which of all others has simulated aneurysm most successfully is the *pulsating sarcoma*, almost always of bone. These growths are so vascular that they pulsate expansilely with each heart-stroke, where they are not contained within a bony shell; they yield a bruit which is described as "soft and whiffing," and sometimes a "thrill"; compression of the artery above causes cessation of these signs, but, as a rule, little or no diminution in size, and on release of the artery there is no increase, merely return of pulsation. When these growths have formed in the line of a great artery, and deeply—as in the iliac fossa—mistakes have occurred through no lack of skill. The signs which specially point to sarcoma are—irregularity of form; possible expansion or perforation of the bone (*e.g.* hip-bone and appearance of pulsation in the buttock); displacement of the artery by the growth (*e.g.* the vessel may be traced over it); expansion less marked than in aneurysm, and perhaps localised at one or more spots; egg-shell crackling; the slight effect of compressing the artery; secondary growths. But it is obvious that the diagnosis from a leaking iliac aneurysm with no reliable history to guide, may be very difficult. Exploration should be borne in mind, but in the iliac fossa of a stout person it would not be an easy matter.

When the ANEURYSM IS SOLID or nearly so, it is motionless, or has only a heaving pulsation. *All aneurysms do not pulsate.* The danger is that the solid mass will be taken for a tumour over the artery. The history is of great importance—first, of a pulsating tumour; next, of its gradual or sudden solidification.

If sudden, there will perhaps be a history of sudden pain (embolism) or of a period of difficulty in the circulation of the part. Lastly, it is important to know whether the mass is enlarging or shrinking. Absence of, or very feeble pulse beyond, in all positions of the limb (*e.g.* with knee flexed), suggests aneurysm strongly; so too does enlargement of collateral vessels, which should be searched for. These are points in favour of aneurysm.

When an aneurysm becomes rapidly and widely diffused, the question arises, whether at the bottom of the swelling there is a ruptured aneurysm or a ruptured artery. The history must, as far as possible, decide. The recognition of a circumscribed arterial hæmatoma, as a rule, presents no difficulties. But certain cases—fortunately very rare—of leaking aneurysm or artery may so closely resemble abscess as to defy diagnostic skill. The state of the peripheral pulse is most important.

When SUPPURATION ensues about a recognised aneurysm, there is no diagnostic difficulty. If there is any pulsation, any disturbance of the peripheral pulse, any suspicion of a non-pulsatile tumour deep in the cellulitis along a great artery, every care must be exercised in taking the history and in treating the case; for not only does the inflammatory swelling tend to obscure the aneurysmal tumour, but an arteritis with secondary thrombosis is often induced. The *treatment* must be that of inflammation generally; and, if suppuration seems inevitable, free aseptic incision into the tissues round the sac should not be delayed. Should serious hæmorrhage occur, the choice of treatment lies between an iodoform plug (when nothing better can be done, as at root of neck); excision or incision of the sac, while the circulation is controlled; aseptic ligature through normal tissues above the aneurysm, no large branch intervening; and amputation, where this is possible. Many consider that if proximal ligature has not been done and suppuration is occurring, the operation should be performed at once with every precaution against infection of the wound.

There are some rare conditions, not included under these four headings, in which diagnostic difficulties arise.

An increasing fluid aneurysm may be pulseless; its history may be a long one, and pulsation may have been absent throughout. Such swellings are taken for cysts or malignant growths, according as the sac is thin or thick. When incised the blood streams out rather than jets. No certain explanation of absence of pulsation under these circumstances has been offered.

A swelling, usually in the neck, having no pulsation, may suddenly increase and pulsate. An artery has probably opened into a chronic abscess.

PROGNOSIS.—This is usually grave. Just as a man who has ruptured one cerebral vessel lives expecting another to burst, so it is with the subject of an aneurysm. Only too often he dies of another, the first having been cured. John Hunter's fourth case (patient æt. thirty-six), however, survived the operation fifty years.

In aneurysms of the subclavian, innominate, upper end of the external iliac, the prognosis becomes grave on account of the aneurysm itself; and the frequency with which cerebral complications have followed upon the treatment of carotid aneurysms is a special point to be borne in mind.

A stationary condition or marked tendency to spontaneous cure will be favourable points.

LOCAL TREATMENT.—Only one method affords a certain obliteration of aneurysm, viz. excision of the sac, or excision with suitable after-treatment. All other methods—which do more than give external support to the sac—aim at so altering the conditions within the sac—by slowing or arresting the flow of blood, by injuring the lining of the sac, or by directly producing coagulation of some blood—as to cause it to fill with clot, as in spontaneous cure; and all may fail in their object.

METHODS.—Elevation, with bandaging. Application of ice. Excision and incision of the sac. Ligature. Compression. Amputation. Needling. Electrolysis. Introduction of foreign bodies. Manipulation. Injection of coagulants. Injection of ergotin.

1. *Elevation, with bandaging.*—In aneurysms—especially popliteal

—trial may be made of the application of a firm bandage over cotton wool to the whole length of the limb, fixation to a splint and vertical suspension of the limb. The mode of action is obvious.

2. *Ice over the Sac* was often employed with rest, etc., especially in recurrent pulsation. It should not be used if the circulation is much embarrassed.

3. *Excision and Incision of the Sac.*

Excision of the sac is preferable to incision with evacuation of contents, because there is much less chance of some branch opening into the sac being left untied to bleed later, and because the raw surface left is more suitable for speedy union and more capable of resisting infection. In cases of diffuse arterial and aneurysmal hæmatoma the sac of the hæmatoma cannot be removed; but, in the latter case, the ruptured aneurysm should be sought at the bottom of the cavity, removed, and the ends of the artery tied. In cases of true aneurysm, or of well-circumscribed hæmatoma, the sac may be so intimately adherent to important parts—especially veins, nerves, and the posterior ligament of the knee—that its complete removal cannot be effected. Then the adherent parts should be scraped free of clot, and the rest removed.

There are three general points to be observed in excision and incision of sacs. The first is to obtain control over the circulation. The next is to make a wound free enough to thoroughly expose the swelling; often a very long one, with division of muscles and even bones. The last is maintenance of asepsis.

The neatest operation is perhaps proximal ligature, separation of the sac and ligature of branches rising, and finally distal ligature of the trunk. The more common course has been to tie the artery above and below, and then to remove the piece between with the sac. When the sac has widely overlapped the trunk, incision of the sac, ligature of the artery through it, and finally careful removal of the whole sac has been done—the result being exactly the same as in excision. In a very serious group of widely diffused aneurysms (femoral, popliteal and posterior tibial), in which after splitting open the hæmatoma the true sac was found and excised, all recovered well.

Results of Excision.—With Walsham's cases, we have thirty-five of spontaneous aneurysm treated by excision of the sac.

	Recovered.	Died.	
1 Carotid,	1	0	
3 Subclavian,	2	1	From another aneurysm.
7 Femoral,	7	0	
22 Popliteal,	20	2	1 suppuration in knee. 1 gangrene, amputation refused.
1 Perforating of profunda,	4	0	
1 Posterior tibial,	1	0	

As to the two deaths among the popliteal cases, that from suppuration in the knee should not have occurred; that from gangrene should probably count only as a case of gangrene, not as one of death. If this is the fair way of regarding the statistics, it must be admitted that in a series of thirty-five cases, grave beyond the average, one case of gangrene directly due to the operation, one of small slough on the foot, and one of small blue spot on each side of two toes, do not constitute a serious indictment of the treatment employed. These were all in popliteal cases.

Results of Incision.—Walsham gives 7 other cases—2 carotid, 2 ilio-femoral, 3 common femoral—treated by incision of the sac, ligature above and below, and plugging. All recovered well: one of the femoral cases had a sharp secondary bleeding.

Wound of the vein is the most likely accident in these operations, and is best avoided by care, and by leaving a strip of sac adherent to it. It was wounded 10 or 11 times in the above 42 cases, and, although the results do not seem to have been so serious as some would have anticipated, it is certainly not a thing to be careless about. No ill followed in the subclavian (1) and femoral (2) cases; but of 7 popliteal cases, 3 showed gangrene or some tendency to it, as above noted.

Delbet contrasts excision and ligature. Using only his modern figures (1888 to 1895), we have 109 cases of ligature of great vessels, 86 of excision of the sac (doubtless including many arterial hæmatomata). The results are :—

	Mortality.	Recurrence.	Gangrene.	After-results.
Ligature,	8·33%	5%	8·25%	Often imperfect.
Excision,	0·00%	0	2·77%	Good.

Against excision it may fairly be urged that the operation is a good deal more difficult than the corresponding ligature ; that it takes a much longer time, and is a decidedly more serious operation for the patient. For excision it is claimed (1) that in spite of its seriousness and extent, it is actually a much safer operation. (2) That there can be no recurrence. (3) That the chance of gangrene is much reduced, because, instead of introducing another obstruction into the circulation of the part, as ligature does, excision removes (unless the vein is injured) an important obstruction both to veins and to collateral arteries, as is shown by the frequent appearance of pulsation in the tibials within fourteen days of an excision. (4) That it removes also a source of embolism and a focus from which thrombosis may spread widely. (5) That removal of the sac rids the patient of the possibility of many after troubles, such as hampering of a joint by a mass of encapsuled fibrin ; pain, anæsthesia, paralysis and trophic changes due to pressure on nerves or involvement of them in the shrinking capsule. (6) That the patient is sooner fit for work.

Theoretically these claims appear to be justified. The statistics available—insufficient as yet—bear them out. It seems probable, therefore, that the operations of excision and incision of the sac will be used more and more in the future.

Meanwhile, other methods of treatment, easier from the practical point of view, give fair results. Excision can always be done in case of recurrence after other methods, and usually when the after-result is unsatisfactory ; it is too late, however, when gangrene has ensued. It must be borne in mind that it is in the more difficult cases of large and diffused aneurysms that excision, rather than ligature, is so essential to save life and limb.

LIGATURE.—A ligature may be applied on the proximal or distal side of an aneurysm. Ligature close above the sac is the operation of Anel; at some distance above the sac, no large branch intervening, it is Hunter's operation; if below the sac, no branch intervening, it is Brasdor's; if branches intervene, it is Wardrop's.

In tying an artery for aneurysm we should try to produce stagnation of blood in the sac, in the hope that being kept at rest in contact with an abnormal surface, it will coagulate, not only in the sac but in the artery whence it springs. To this end, the nearer the thread is placed to the aneurysm the better; for when an obstruction arises in the main trunk, the branches rising above it and neighbouring vessels dilate and convey blood past the obstruction into the main trunk again below the obstruction, and thus a stream of blood sufficiently swift to prevent clotting may be kept up through the sac. Ligature even immediately above the sac will not ensure complete quiescence if branches arise from the sac, and under some other circumstances. For a long time it was believed that the object of ligature was to reduce (not stop) the flow through the sac, so as to favour the formation of firm white laminated clot; but cure of an aneurysm after ligature usually takes place too quickly to allow of the deposit of laminated fibrin. Inflammation does not result from rapid coagulation of blood in a sac.

Up to 1785 popliteal aneurysm was treated by amputation, or by incision of the sac. The results of the latter were most disastrous under the then conditions; and, having recently lost a case, John Hunter asked himself, "Why not tie it up higher in the sound parts, where it is tied in amputation, and preserve the limb?" Hunter operated in Hunter's canal in five cases, with four recoveries.

Similar success was achieved by others, and this and the relative ease of the operation caused it to become rapidly established. Scarpa shifted the seat of election to a higher and still more accessible point, noting that no branch of importance arose between.

It thus became established that, in a large number of cases of aneurysm, cure could be effected by proximal ligature at a distant point, provided no large branch arose between; but even this is not a certain bar to cure.

Mode of Cure.—There is only one, viz. the obliteration of the sac and of the artery whence it springs by clot, and later by their conversion into fibrous tissue. In the majority of cases clotting in the sac and artery is rapid, and the clot, doubtless, red. The artery is obliterated at two points after Hunter's operation: at the seat of ligature and at the aneurysm, the artery between being usually patent. The two obstructions run together after Anel's operation.

Clinical Effects of Ligature.—As the thread is tightened, pulsation in the sac and distal vessels ceases; the sac shrinks considerably; the limb becomes pale; its temperature tends to fall; and it feels heavy and dead. In the course of a few hours the limb is often hyperæmic, all its vessels being dilated as the direct and reflex effects of the anæmia, and in this state its surface temperature may be higher than that of the normal limb. Soon superficial arteries, not previously noticeable,—*e.g.* those around the patella,—can be felt enlarged to carry on the collateral circulation. The hyperæmic state does not last many hours; the temperature then falls to below that of the other limb, and nutrition generally remains more or less subnormal. The sac usually hardens within twenty-four hours, and steadily becomes smaller and harder. After three or four weeks it should be quite hard, and much reduced in size, though it takes a long time to complete the absorption of a large mass of old clot. The patient may begin to get about, as a rule, in a month.

Treatment.—After ligature of a main trunk the limb should be wrapped in a thick layer of cotton wool, placed with its distal extremity slightly raised to favour venous return, and hot bottles should be put under the clothes around, but not in contact, with the limb. The finger-tips or toes should be easily exposed, and their colour and circulation should be often examined. Increasing congestion and œdema demand somewhat greater elevation of the foot, and long-continued upward massage.

Success has followed proximal ligature (aseptic) in about 86 per cent of all cases, failure in 10 per cent, death in 3 per cent, gangrene in 10 per cent.

Accidents after Ligature—Secondary Hæmorrhage.—This should be treated by re-ligature in the wound, the vessel being traced up and down to beyond the region of septic arteritis, every attempt being made to render the wound aseptic. Proximal ligature, hard by but through healthy tissues, may succeed. Amputation at the seat of ligature is the final remedy in limbs.

Suppuration of the Sac: generally, but not always, connected with a septic ligature in its neighbourhood. It is most common in the neck, axilla, and groin. Treatment—that of cellulitis; incision, aseptic and free, as soon as pus is detected—too soon rather than too late. If the sac suppurates, incise and plug, or excise. Hæmorrhage may be dealt with by excision and ligature, unless the part be greatly damaged by the suppuration and life threatened; then amputate above the aneurysm.

Recurrent pulsation is due (1) to some imperfection in tying the ligature. This may not have been tied sufficiently tightly to occlude the vessel, either on account of clumsiness of the ligature or of over-anxiety to avoid rupturing the inner coats, or because the pulsation of the artery has loosened the first knot before the second was added; or the knot may have come undone soon after closure of the wound. (2) To imperfection in the ligature rendering it liable to too rapid absorption. This happened with some early specimens of catgut. (3) To the rapid establishment of a too free collateral circulation, so that blood in a brisk stream soon reaches the main trunk below the ligature. This is the common cause.

Recurrent pulsation is a common phenomenon during the first few days after proximal ligature. Rarely it occurs months or years after supposed cure. It varies in strength from the slightest perceptible movement, which may gradually increase to pulsation as strong as before the operation. Usually it dies away, or if it does not, is easily stopped by simple treatment. It may persist. The aneurysm of which it is the seat is often much smaller and firmer, but when the patient gets about it will yield slowly or rapidly.

Treatment.—Should the pulsation not disappear during the period of healing of the wound and recovery from the operation, it should, if slight, be treated by still further diminishing the supply to the sac by elevation, bandaging, or compression. If marked, a careful consideration of its cause is necessary. If the artery is pulsating at the seat of ligature the sooner it is re-ligatured the better; but it is not always possible to be sure of this. The nature of the ligature, the strength of the pulsation, the effect of compression between the ligature and the sac, the effect of compression of great collateral branches and of the main trunk beyond the sac should all be tried, and the most probable conclusion drawn. When the artery has been tied immediately above the sac (*e.g.* end of ext. iliac for common femoral aneurysm), the wound should be reopened, if there is any reason to suspect the ligature, and the vessel re-tied. If the ligature is not at fault in a case such as this, or in axillary or subclavian aneurysm, the choice would lie between seeking and tying the most likely collateral branches, *e.g.* epigastric and deep circumflex iliac, and excising the sac. In the case of the subclavian or axillary an operation to find the collaterals would probably be more difficult and less satisfactory than that for excision. In other cases where there is more room, if the first ligature is not at fault, tie again close to the sac—*e.g.* after ligature in Scarpa's space, tie below the anastomotica magna—or excise the sac. But if the femoral is pulsating, where tied, repeat the ligature, if only to make sure that the artery has not been injured.

Another form of failure to cure is rare. The contents may remain fluid without pulsation, and the sac may even enlarge and burst after proximal ligature. Regurgitant blood-supply has been suggested as the explanation. Excision of the sac is the proper treatment.

Gangrene.—The obstruction to the circulation caused by an aneurysm may alone cause gangrene. When we add to this an obstruction higher up on the same main trunk, and when we consider that cardiac weakness and disease, and widespread arterio-sclerosis are not uncommon in subjects of aneurysm, we can feel no surprise at the occasional occurrence of gangrene. Accidents at the operation, such as serious loss of blood, injury of the vein, infection and cellulitis, greatly increase the danger of gangrene; while treatment of a ruptured aneurysm by Hunterian ligature renders it still more likely.

Gangrene is usually obvious between the third and seventh days, but may be earlier if impending before operation, or later when due to spreading thrombosis or to embolism.

Both moist and dry gangrene occur in connection with ligature. Moist gangrene is usually the more extensive, and is infinitely the more serious; but dry gangrene sometimes involves a whole leg, and is, upon the whole, the more frequent variety. It may cause a mere scab.

Treatment.—In dry gangrene await a line of demarcation. In moist gangrene amputate above the aneurysm. When gangrene is threatening, proximal ligature at a distance should not be practised; excision of the sac or ligature immediately above the sac can alone be considered. The former seems to be the better operation. It, or incision, should be considered whenever gangrene threatens after a Hunterian operation.

Late Results.—After successful proximal ligature aneurysms steadily shrink, their pressure symptoms disappear, and in the course of a few weeks or months the patients return to work without inconvenience. Not very uncommonly recovery remains incomplete and the patient more or less unfit for work; there is more or less œdema; the part is weak, cold, wasted, liable to ulcers; it is the seat of numbness, of more or less severe pain, and even of trophic lesions, these latter symptoms occurring especially in connection with aneurysms pressing on the brachial plexus.

Treatment.—Amputation has occasionally been done for the relief of these—usually upon some mistaken diagnosis. Excision of the shrunken sac has given relief, and should always be tried.

PROXIMAL LIGATURE CLOSE TO THE SAC, no branch intervening, places the obstruction to the circulation as low as possible, and reduces it almost to that of the aneurysm alone; gangrene will therefore be less likely to ensue. The collateral circulation will be employed only in conveying blood past the aneurysm, not into that portion of the artery between ligature and sac which usually remains pervious. This is the highway to the sac, and, with it open, failure to cure is more likely than when only regurgitation or small muscular and articular currents are possible. Accordingly, recurrent pulsation would probably be less common in proportion as the ligature approaches the sac. Possibly mechanical irritation in the immediate neighbourhood of the sac would tend to excite clotting. Objections are: (1) Greater difficulty: under ordinary circumstances the difficulty certainly is not great. If the aneurysm encroaches on the upper part, say, of the popliteal artery, the end of the femoral is the proper spot for the ligature. (2) The vessel is likely to be diseased so close to the sac: this is not the case.

Conclusion:—The success of proximal ligature is likely to increase in proportion to its proximity to the sac.

DISTAL LIGATURE.—This is far less efficacious than proximal ligature, and Brasdor's operation, with no branch intervening, is the most successful form. Unfortunately, except in aneurysm of the root of the carotid, it is difficult to find a place where it is theoretically possible. Brasdor's operation acts as an embolus would. The blood is brought to a standstill in the sac, collateral vessels dilate, and pressure is taken off. Some suppose that coagulation starts at the ligature and spreads back into the sac.

Wardrop's operation has found its most frequent application in cases of aneurysm at the root of the neck.

Accidents.—Enlargement of the sac has sometimes occurred; also suppuration of the sac.

COMPRESSION.—(1) *Directly upon the sac;* often caused sloughing and disastrous results. It is now used chiefly as a means of support by a bandage, simple or elastic, or by a moulded and padded cap.

(2) *Proximal compression,* digital or instrumental. *Object:* diminution of flow through an aneurysm, so that clotting may take place while the

artery remains pervious at the point compressed. *Digital compression.*—For this relays of assistants are required. The patient should be placed as comfortably as possible; the part where pressure is to be made should be shaved and powdered. Two spots, one higher, one lower, for the thumbs should be indelibly marked. The thumbs should be placed on the artery, sparing the vein as much as possible, one on top of the other, so that the actual pressure may be made with either. A conical tense shotbag of four pounds may be allowed to rest on the thumb. The pressure required is just enough to stop pulsation. At each change of relays—every hour—pulsation should be cautiously felt for. Arteries suitable for digital compression are the femoral at the groin, common carotid, subclavian, and brachial, where these vessels lie on bone and are fairly superficial.

Instrumental compression is not so well borne as digital, but requires fewer assistants. It is used only in aneurysms of the lower limb. Carte's instruments for the ext. iliac and femoral are usually employed. They are used alternately, just like the thumbs of assistants. Lister's tourniquet has been used in aortic and iliac aneurysms. If the patient is restless and irritable he is kept under morphia; in some cases chloroform is given lightly.

Compression may be *continuous* for 12-24 hours or longer; or *discontinuous*, being carried on for several hours daily over weeks, or even months. It is best, when possible, to make an attempt to cure a case straight off by continuous complete control of the main trunk, remembering that the longer success is postponed the less likely is it to come at all.

The *mode of cure* is the usual one. When it occurs gradually the fibrin is laminated; in this case the sac slowly hardens, pulsates less forcibly and with less expansion. Often the cure is quite sudden, even in the longer cases. Again, a sac may harden and soften once or twice before cure is effected. It is necessary to protect the clot in the artery from the full force of the heart for two or three days by the light action of a compressor. Relapse may occur long after apparent cure, the artery, presumably, not being obliterated.

Advantages claimed for compression were that it avoided "an operation"; that the dangers incidental to a wound in which a great artery was tied were not run; that gangrene was less likely than after ligature, because the artery was blocked only opposite the aneurysm; that compression was likely to throw less strain than ligature on the great central arteries; that ligature could be practised with less likelihood of gangrene after failure of compression, because the collateral circulation would be developed more or less. The last two points are at least doubtful. The *objections* are:—That it is always painful and wearisome, and may be too painful for some temperaments to bear: morphia, or light anæsthesia, can, however, usually be employed; that several skilled assistants are required for digital compression, which is certainly best; and that long compression may render subsequent ligature more difficult (from matting, if it has to be done at the point of pressure, which generally can be avoided) and less successful, owing to development of the collateral circulation.

The palmy days of compression were those of septic surgery. The reports of three London hospitals do not show that compression has been employed at all in their wards since 1891. The dangers of sepsis have been all but abolished in ligature wounds. Ligature is more certain to succeed, and, if not used when excision should be done, accidents are not more likely than after compression. The wounds are usually painless. Under these circumstances ligature has come more and more to the front as the method of dealing with aneurysm *cito, tuto et jucunde*. The places where the ligature is still in difficulties, *e.g.* first part of subclavian and innominate, are just those where compression can afford no help.

The *success* of compression amounted to quite 50 per cent in popliteal aneurysms; less in other varieties.

Accidents were not infrequent; but compression was used in cases of bad heart disease, general arterio-sclerosis, and in other conditions in which operation was undesirable. The accidents were: sloughing and soreness at the point of compression, gangrene after solidification of the sac and suppuration of the sac; both aneurysm and arterio-venous fistula have been recorded at the point of pressure.

In cases where it is really desirable to avoid a wound, compression may render good service. Like ligature, it should be most effectual near the sac, yet not so near as to injure it. Unfortunately the points at which compression is possible are few and fixed.

Flexion of the knee and elbow, especially over a pad, stops the pulse in the distal arteries. It is sometimes used as a means of compression, being kept up for hours or days. Its success has not been marked, and it has often been abandoned on account of pain.

Distal compression may be usefully combined with the proximal when the collateral circulation is free. It is sometimes used alone under the circumstances in which distal ligature is employed, but is a poor remedy.

COMPRESSION BY ESMARCH'S BANDAGE, suggested by Reid as a means of inducing in aneurysms that absolute stagnation which so greatly favours coagulation. A rubber bandage is wound firmly round the *horizontal* limb up to the aneurysm, lightly over the sac, and again sufficiently firmly above it to entirely control the blood-supply and to keep the sac comfortably full of blood; when the aneurysm is high in the limb there may not be room even for an elastic cord above it. It must then be combined with digital or instrumental compression above the sac The sac should not be left quite unsupported, as it may fill even to rupture from the surrounding parts.

The bandage should be kept on for one and a half hours, and not longer. It causes much pain, necessitating morphia freely or light anæsthesia. The artery above the sac must be controlled before the rubber bandage is removed, and either light digital or instrumental compression, to prevent disturbance of young clot, kept up for two or three days. On removing the bandage the aneurysm may be cured, *in statu quo ante*, or in any intermediary stage. According to Gould it succeeds in about fifty per cent of the cases. A second, or even a third attempt may be successful; but 67·6 of all successful cases occurred after one application.

The *mode of cure* is by rapid coagulation in sac and artery.

Causes of failure.—Too brief duration of stasis; a fusiform or small saccular sac with wide mouth and fairly normal endothelium; too early exposure of the fresh clot to the heart force.

Advantages of Reid's method: it acts quickly if at all; it is widely applicable; it causes no wound; it interferes with no subsequent treatment should it fail. *Objections:* It may so raise the blood pressure as to injure some morbid central vessel or internal aneurysm. It may cause gangrene: this occurred only twice in sixty-five cases, and in neither did the bandage seem to be wholly responsible. It may rupture the sac; this should be supported by the bandage. Rupture occurred only once in seventy-two cases. In another case the sac was much larger. In spite of these apparently good results in recorded cases, the method is regarded with distrust and is not now much employed.

AMPUTATION at the shoulder has been practised a few times in subclavian aneurysm, with the idea of diminishing the flow of blood through the subclavian by removing the part to be supplied. Only partial success in one case has been recorded.

NEEDLING.—In 1890, Macewen advocated the treatment of aneurysm by the aseptic introduction into the sac of fine needles which were to act by gently and superficially scratching and irritating the sac-wall. One or more long, fine pins are to be passed in until they just touch, and no more, the opposite wall. Here they are gently moved so as to effect the scratching, or the pulsations are allowed to do so; after ten minutes or so they are shifted to another spot and a considerable area is worked over methodically. The needles are introduced at different points to reach the whole inner surface. They are left in some hours, but never longer than forty-eight. Several pins should not be introduced close together lest the wall be too much weakened. Specially thin points in the sac should first be attacked. Whilst the pins are in they are surrounded with wet gauze, and an antiseptic dressing is kept on for some days after the operation. As a rule distinct thickening of the wall where scratched is recognisable early. The sittings are repeated at suitable intervals.

Macewen's object in this treatment is to induce the formulation of white thrombi over the area scratched, and he endeavours thus to act upon the whole interior of the sac. He quoted four cases in illustration of the method, and it would seem that needling is worthy of a more extended trial than it has received.

ELECTROLYSIS has sometimes seemed to retard the progress of an aneurysm; those who have used it largely claim decided improvement in over half the cases treated—chiefly aortic aneurysms. But it has been used successfully also in combination with compression to induce coagulation in external aneurysms. Around the positive pole a fairly firm clot forms, around the negative a loose mass, frothy from liberated hydrogen and useless as a means of consolidating the sac. Much difference of opinion exists as to how electricity may be used to the greatest advantage in these cases. (See Electrolysis.)

Embolism would seem a likely danger, but is not known to have occurred. Sloughing round the punctures, oozing and hæmorrhage, infection and cellulitis, have been pretty common.

INTRODUCTION OF FOREIGN BODIES INTO THE SAC.—This was suggested and carried out by the late C. H. Moore in a case of thoracic and otherwise inoperable aneurysm. Since then wire of various kinds, horsehair, fishing gut, and watch-springs have been introduced through a fine cannula passed in obliquely through a well-covered portion of the sac. Considerable loss of blood may occur through the cannula. Every care to preserve asepsis must be employed. The wire has generally been pushed in until some resistance has been met with, then cut, and the end completely sunk.

The success of the method has been so slight that it is doubtful whether it should be practised.

MANIPULATION.—Sir William Fergusson practised this in two cases with the idea of squeezing fibrin from the sac-wall into the distal orifice of subclavian aneurysm. Having emptied the sac by thumb-pressure he squeezed and rubbed its side somewhat. In the first case giddiness resulted, and after one or two repetitions of the manipulation, pulsation ceased and the sac shrank; but it burst seven months later. In the second case the distal artery was obliterated, and manipulation was followed by hemiplegia; but the sac shrank gradually, and the man was alive and well two years after. Many aneurysms have become solid, either at once or within a day or two after handling, sometimes from embolism, sometimes probably from some inflammatory change in the sac. Embolism is, however, a dangerous complication, and the method has been very little used.

The *INJECTION OF COAGULANTS.*—The danger of embolism is so great and suppuration has so frequently ensued after injection of perchloride of iron (Pravaz) drop by drop from a screw syringe, that this method should not be employed. A solution of fibrin-ferment has been tried without success, and Wooldridge's ferment appears to be too powerful to be safe.

The *Injection of Ergotin* around the sac is useless, and acts only as an irritant.

Intra-mural Hæmatoma—Dissecting Aneurysm

When owing to some lesion of its inner surface, blood finds its way into the substance of the wall of the aorta, it "dissects" the outer from the inner layers as it forces its way onwards.

Seat.—Primarily, almost confined to the aorta; secondarily, the hæmatoma may extend along any or several of the primary branches. In one case it extended from the ascending aorta to the left popliteal. It may start in other great arteries, *e.g.* iliacs, as in Oliver's case—in which there were four separate lesions in the ascending and descending aortæ and both iliacs. The rupture of the inner coats is usually in the ascending aorta (55 out of 73 cases), but it may be as low as the bifurcation. The tear is usually transverse in the ascending aorta, and may extend all round the vessel: usually it runs only about half-way round. In the descending aorta the tear is more commonly vertical. From the initial rupture the blood works its way in all directions in the substance of the middle coat. In the great majority of cases a second rupture soon occurs through the external coat, and blood is extravasated into a neighbouring cavity surrounding connective tissue or adherent viscus. In 8 or 10 per cent of all cases the second rupture occurs through the internal coats back into the aorta; the outer layer thickens, and life may be considerably prolonged. The appearance on section, in such cases, is that of one tube within another, touching nowhere or along a line of varying width at different levels. When this line widens to something like half the circumference the appearance of the section is like that of a double aorta. Blood flows through the new channel, which acquires a smooth aspect and is said to be lined by endothelium. In cavities where there is no flow the blood coagulates, and red or white clot is found. Where the dissecting process passes the origin of vessels it tends to extend along the trunks. The smaller (*e.g.* intercostal) are often torn across, even the larger are often thrombosed. Vessels thus affected are usually highly atheromatous; chronic interstitial nephritis, aortic disease, and hypertrophy of the ventricle are common. But, exceptionally, all these may be absent.

Etiology.—Dissecting aneurysm appears to be a little commoner in males than females, and occurs in middle or advanced life. Sometimes a violent or distinct effort starts the trouble; but often the patient is sitting or standing quietly, doing nothing to raise the blood pressure. This is often high, however, from renal or cardiac disease. The marked atheroma, which is usual, renders rupture easier; but in some cases there has been little or none. Then congenital weakness, fatty degeneration of the middle coat, perhaps connected with obstruction of a vas vasis and pure trauma, are the explanations offered.

Symptoms.—In the acute cases—sudden severe pain, usually in the thorax, sometimes in the loins, and collapse, rapidly fatal or recovered from until the complete rupture occurs.

In the chronic variety the train of symptoms is as follows:—sudden severe pain, often causing fainting (but sometimes no such pain occurs, as in Oliver's case). The pain is said not to radiate down the arm, and not to cause sense of constriction of the chest. Gradually the patient recovers and returns to work. Then follow symptoms as of heart disease—œdema of the legs, dyspnœa, palpitation. The pain may recur now and again.

Usually a rough systolic murmur is heard over the cardiac area and on left of spine. In such cases life has been prolonged even to eight years, external rupture or cardiac failure being the immediate cause of death.

Anuria may result from blocking of both renal arteries. Tearing or blocking of several lumbar arteries leads to sudden, perhaps temporary, paraplegia. Blocking of the innominate may cause hemiplegia. Absence of symptoms, or presence of such as have just been noticed, usually throws the observer off the scent.

Treatment can be only symptomatic whether a diagnosis is made or not.

ARTERIAL HÆMATOMA

When an artery is wounded, and there is some obstacle to the free escape of blood, it accumulates around the artery and forces its way along the neighbouring areolar planes, forming a hæmatoma or blood-tumour. Under certain conditions this swelling may exhibit pulsation, bruit, thrill, and other signs of aneurysm—hence it is often called a "traumatic aneurysm." A similar state of matters results when an undilated artery is ruptured subcutaneously, or wounded by a fragment in a fracture. The obstacle to the escape of blood upon the surface is the only difference between these cases and corresponding injuries of arteries in open wounds. We have seen that similar hæmatomata may arise from the leaking or rupturing of an aneurysm.

When a wound in a large artery is quite small, longitudinal or punctured, and pressure is well applied, it will heal; but yielding of the scar may occur, and a true traumatic aneurysm manifests itself as the extravasated blood is absorbed. It will have a sac from the beginning continuous with the arterial coats.

But when a narrow, long, oblique wound of the superficial parts leads to a considerable wound in a main artery, and when any pressure applied does not close the wound in the artery, a hæmatoma will form. Its size will vary with the amount and duration of collapse, the magnitude of the artery wounded, the size and direction of the wound in it, and the laxity of the surrounding tissue and the efficiency of any pressure employed. The extreme results are—(1) Such widespread extravasation into the part that the patient shows the effects of hæmorrhage, whilst, locally, the circulation is increasingly impeded by the wound and pressure of the hæmatoma, until gangrene supervenes (for Symptoms, see p. 221); and (2) the rapid development of a swelling of no great size, and fairly outlined from the first, in which all the signs of aneurysm are present. Every stage between these extremes may be met with: the more diffuse the hæmatoma the more marked will be the signs of circulatory difficulty, the less marked will be the signs of aneurysm; but these appear as the hæmatoma becomes circumscribed, by a capsule of connective tissue and clot, while the marginal portion of the extravasation is absorbed. Pulsation and bruit are best marked in cases of considerable transverse, lateral wound; least marked with a small longitudinal wound or a complete division allowing the ends of the artery to retract within the sheath. Ultimately a circumscribed hæmatoma may differ from an aneurysm only in its etiology. If untreated, it tends to rupture.

Sometimes a hæmatoma will reach a great size without threatening the vitality of the part, *i.e.* its expansion is satisfied by the yielding of parts towards the surface; it may rupture here. It may also inflame and suppurate. With regard to size, it is much more astonishing to see how frequently a wound of a great artery leads to a circumscribed hæmatoma than to find it giving rise to a diffuse one.

Diagnosis.—From cellulitis and deep abscess, and from ruptured aneurysm, depends largely in the first case and entirely in the second upon the history.

From malignant growth. This may sometimes arise. There is a history of a blow or injury to a limb, swelling follows and increases steadily; there is a good deal of pain. The swelling is fairly outlined, elastic, not fixed to bone. No sign of bruising and none of inflammation. An incision may be necessary to make the diagnosis.

Treatment.—The well-circumscribed form may, like the aneurysm it so closely resembles, be successfully treated by compression or proximal ligature; it is equally true that these remedies may fail. The only certain cure is excision or incision of the sac; and, seeing that the vessel is presumably healthy, there can be no question as to the propriety of tying it as one would any other wounded artery.

But, if proximal ligature is justifiable in cases of circumscribed hæmatoma, it is a most dangerous and inefficacious practice in the diffuse form in which the circulation is already much impeded. Here control of the circulation, while the hæmatoma is split open freely, the wounded vessel found and its ends tied, is the proper treatment.

Arterio-venous Inter-communications

Causes.—In the great majority of cases these are of *traumatic* origin, being due to stabs and cuts, not infrequently to gunshot wounds, rarely to contusions without wound, involving an adjacent artery and vein. Sometimes there has been no injury, the communication resulting from some disease (*spontaneous*). Bramann's examination of these cases shows that they were due either to a wound which had been overlooked or to perforation of a vein by an aneurysm. There does not, however, seem good reason for denying that the base of an atheromatous ulcer in an undilated artery may adhere to a vein, and, not finding sufficient support, may open into the vein—the opening, perhaps, being brought about by some fall or strain; indeed, this rather than pressure atrophy, probably explains the opening of a true aneurysm into a vein.

Seats.—In Bramann's table venesection at the bend of the elbow was responsible for half the number of cases collected; a few occurred after temporal arteriotomy. The distribution of the remainder shows that these lesions may occur wherever an artery and vein are liable to simultaneous injury, no matter what the magnitude of the vessels. Thus Colzi (*B. M. J.* 1895, vol. ii. p. 184) has recorded a case of direct communication between the aortic arch and the left innominate vein, the result of a stiletto wound; the man lived for eleven years. This is probably unique. The carotid and jugular in the neck, the carotid and cavernous sinus ("pulsating exophthalmos," "orbital aneurysm,"), the axillary and common femoral vessels are the great vessels most often affected after the brachial.

Pathogeny and Morbid Anatomy.—When an artery and vein are wounded contact is occasionally maintained, the edges of the wound unite, and a *direct* communication between the artery and vein results. Still more rarely the communication is *indirect*, being effected by an irregular, more or less spherical cavity opening at either pole into one or other vessel. This indirect communication is often called a varicose aneurysm, because the fibrous sac between the vessels feels and pulsates like an aneurysm. In this sac there is, as a rule, no clot.

As results of the escape of blood from an artery into a vein we find: that the parts beyond suffer in their nutrition, the muscles degenerate and waste; that the veins dilate greatly opposite the opening and for long distances both up and down, the unsupported superficial veins suffering more than the deep. They lengthen, also, and become tortuous. The dilatation is accompanied by great hypertrophy of the vein wall. The venules leak, the subcutaneous and other connective tissue becomes œdematous and thickened. The arteries beyond the opening contract.

Symptoms and Signs.—These may be best shown by a typical case described by Bramann. A man was stabbed in the axilla; bled very freely, but stopped it himself by pressure, and it did not recur. His arm swelled greatly, the forearm and hand becoming bluish. As the swelling subsided, in the fourth week the patient felt a pulsating swelling in his axilla, which increased gradually until the fourteenth week, when the man tried to work; then it and the swelling of the arm increased.

On admission the left forearm was 3 to 6 cm. too large, cool (always 3°-5° lower), and bluish. Sensation in the hand was lowered, the grasp much weakened. Subcutaneous veins of forearm and inside of arm much dilated and tortuous. In the axilla a 4½ cm. scar just below level of head of humerus; beneath it a hen's-egg-sized swelling raised and thinned the skin. The swelling was soft, compressible, obviously pulsating, continuous with a much dilated vein above and below. There was a strong continuous thrill, increased with each systole; both pulsation and thrill were felt along the large veins above the clavicle and in the lower arm. A loud bruit increased with systole started in the swelling, and was heard in the neck and even in the palm. The pulse below the swelling was weak and delayed. Compression of the subclavian stopped all signs, and the axillary swelling and veins collapsed. Pressure on the main vessels below the swelling caused it to become dangerously tense. Elevation to the horizontal caused the veins to empty. Pressure through the swelling towards the humerus insufficient to close the artery, stopped the venous phenomena, and caused improvement of pulses at the wrist. The patient complained of weakness and sense of weight in the part, rendering it useless for work.

Mutatis mutandis, this description applies wherever the arterio-venous communication may be. If it is *indirect* we must add to it the presence, deep to the chief dilatation of the vein, of a firm, irregularly spherical, pulsating sac, deep to which again is the artery.

The symptoms and signs may be more or less marked in different cases according to the size, depth, and other circumstances of the communication.

The constant pulsation and thrill, and in many cases the constant bruit, are sources of the greatest annoyance to the patient. The bruit is generally a very loud one, but of course varies in quality. William Hunter, who first recognised the true nature of this trouble, compared it to a long, whispered "r."

Diagnosis.—There can be no difficulty except in deep cases with little or no superficial varix. The recognition of a continuous thrill and murmur, with systolic increase, usually conducted both centrally and peripherally, will make the diagnosis; a venous pulse is an equally important sign. To distinguish between the direct and indirect communications a firm tumour, never disappearing, deep to the venous swelling, must be recognised. It varies from a nut to a fist in size, being usually quite moderate.

"Cirsoid Aneurysm" is distinguished from arterio-venous fistula by dilatation not only of the trunk, but also of all the branches of the trunk upon which the aneurysm is situate. Thrill is slight if present. The bruit is feeble, or is produced only by light pressure, is not increased by systole. The pulsation is uniform over the whole swelling, and does not cease but gets weaker on compressing the main trunk.

Prognosis.—This is graver in the indirect than in the direct form; for, although it sometimes remains stationary for a long period, the circumscribed hæmatoma between the vessels tends, like all arterial hæmatomata, to rupture. This form must, accordingly, be treated when recognised. The arterio-venous fistula soon reaches its full development; but its consequences, as shown in the above case, may demand treatment. Further, it may increase steadily. When the brain is included in the area of malnutrition serious cerebral symptoms are likely sooner or later to arise, *e.g.* after eleven years Colzi's patient (p. 234) died of jugular thrombosis and acute cerebral œdema.

Treatment.—In the direct form an elastic support may be sufficient, perhaps with a special pad over the opening. Should this not be enough the proper treatment both for this and for the indirect communication is to cut down and tie the artery and vein above and below the opening, either going through the venous dilatation, or by the side of it as may be most convenient, and then excising the communication. These operations are often difficult and dangerous where the circulation cannot be well controlled; it is therefore necessary in such case to weigh the matter well before operating.

Aneurysms of the Upper Limb

These, taken all together, are very much less common than those of the lower extremity.

Causes.—Up to the lower third of the brachial the great majority are not true aneurysms, but arterial hæmatomata—the results of cuts, punctures, and lacerations. A few cases result from contusions (single, or slight but often repeated) and violent wrenches. The great majority of apparently spontaneous aneurysms below the axillary are due to embolism.

Seats.—The superficial palmar arch, the forearm vessels above the wrist, and the brachial at the bend of the elbow, are the vessels which most often give rise to arterial hæmatomata; but these may occur upon any of the vessels of the part—even the collateral digitals, the deep palmar arch and its branches. Arterio-venous communications have been described in the hand, are more frequent in the forearm, and the bend of the elbow has hitherto been their *lieu d'élection.* Embolic aneurysms are probably most common about the bifurcation of the brachial and the origin of the common interosseous trunk from the ulnar.

Symptoms.—These aneurysms are usually of small or moderate size in proportion to the artery with which they are connected.

Treatment.—Proximal compression, digital or by flexion, may be tried; and vertical suspension is applicable here. These failing, excision of the sac is the proper treatment. The only local consideration which tells against this is great depth of the swelling among important parts—particularly in connection with the deep palmar arch. The free anastomosis in the hand renders proximal ligature specially unreliable; yet ligature of the ulnar and radial above the wrist should be tried here after failure of non-operative measures. Recurrent pulsation may still be overcome by compression directed to the median artery and the anterior and posterior interosseous vessels as they cross the backs of the lower ends of the forearm bones. High aneurysms of the ulnar can be reached without serious injury to the flexor sublimis.

Axillary Aneurysm.—*Causes.*—Wounds and subcutaneous lacerations, usually connected with the occurrence or reduction of dislocations—especially old unreduced—have been responsible for many so-called "aneurysms" in this region. Sharp movements without violence, *e.g.* trying to prevent a hat blowing off, may cause sufficient injury to be followed by aneurysm, and it is probable that the frequent strains thrown upon the artery in the very free movements of the shoulder-joint play a causal part in rendering true aneurysms of this vessel somewhat frequent, especially in men, and on the right side.

Arterio-venous communications are rare.

Seat.—True aneurysm may affect any part of the main trunk. Hæmatomata sometimes spring from the subscapular or other great branch.

Symptoms.—Stationary aneurysms and spontaneous cures are very rare. As a rule these aneurysms grow fast, rupture early, and cause great pain. They grow fast because they lie in loose tissue; nevertheless the axillary sheath and the strong fascia of the floor of the axilla usually direct the enlargement upwards beneath the clavicle into the subclavian fossa. The arm is pushed from the side, the shoulder raised, the axilla distended. The vein is often severely compressed. The nerves of the brachial plexus are either pushed outwards or surround and adhere to the sac, according to its level. Sometimes one, sometimes all the trunks suffer; pain may be extreme, paralysis absolute. If the pulse beyond is not enfeebled and delayed, it may be evidence in a traumatic case that a branch, and not the main trunk, has been wounded.

If unchecked the swelling increases and may rupture subcutaneously, externally, into the shoulder-joint, or into the thorax, often after eroding the clavicle, humerus, or ribs; the latter may fracture as a result. Suppuration is commoner here than elsewhere; gangrene is rare.

Diagnosis.—The principal difficulty is to distinguish a suppurating aneurysm from simple cellulitis.

Treatment.—Digital compression of the subclavian may be tried if the subclavian space is not encroached upon. It may be combined with an Esmarch's band on the limb up to the shoulder. It is best conducted under anæsthesia, and should be kept up for one and a half hours, followed by compression without the Esmarch for at least as long again. Needling may be combined with this. If the clavicle is much raised compression might be carried out aseptically by a rubber-sheathed finger through a suitable supra-clavicular wound.

If this fail, the choice lies between ligature of the axillary close above the sac in low aneurysms, or of the third part of the subclavian in high aneurysms, and excision of the sac. Failure to cure of the usual remedies must be met after trial by excision of the sac.

Opinion seems to be growing in favour of excision as the operation of choice (see p. 224). To excise, control the subclavian either by finger pressure or by a loop passed under it, upon which gentle traction can be made as required (Nélaton, Treves). The incisions must give free access. The great pectoral must be divided, if necessary. Until ready to deal with the distal end of the artery an elastic bandage should be kept round the limb to as high a level as possible. The first thing is to find, clear, and tie the artery above and below the wound, or sac-mouth; then to remove all clot and as much sac as possible, having regard to the state of the patient, and the safety of veins and important nerves. Twice, at least, pieces of the adherent axillary vein have been removed without ill-result. On the other hand, the *results of ligature* of the third part of the subclavian have improved greatly of late years. Suppuration of the sac, formerly so common, septic pleurisy, and pneumonia, formerly attributed to injury to phrenic fibrils in the nerve to the subclavius, and secondary hæmorrhage, have all become rare. Gangrene after ligatures in the upper limb was never common. This improvement in the results obtained by ligature, and knowledge that serious suppuration and bleeding may follow upon excision if the wound is infected, makes a decision very difficult. The case may be put thus:—In small circumscribed aneurysms proximal ligature as near the sac as possible will probably succeed. Here excision would be easy, and more certain to cure and leave a good result. In bad cases with impeded circulation, or with marked evidence of pressure upon nerves, excision, though difficult, gives the best chance of preserving life and limb, and of removing œdema, weakness, pain or paralysis. In hæmatomata the old operation may show that a branch, not the trunk, has been wounded. In recurrent pulsation after proximal ligature resisting simple treatment, excision is easier than the discovery and ligature of collateral branches—and it is certain. In arterio-venous communications there is no other cure.

SUBCLAVIAN ANEURYSM.—Causes.—Subclavian aneurysms are said to occur somewhat less frequently than carotid. Like axillary aneurysms they are much commoner in men than in women, and at least 70 per cent occur upon the right side—both facts pointing to the influence of strain. So well is the vessel protected from direct injury, and so commonly does wound end in death from hæmorrhage, that there are few instances of arterial hæmatoma on record. Arterial hæmatoma or traumatic aneurysm has in rare instances followed quickly upon fracture of the clavicle. By some, fracture of the first rib is regarded as a possible cause. In the great majority of cases there is no history of direct injury. In no fewer than five instances has aneurysm supervened in cases of cervical rib over which the subclavian passed.

Course and Symptoms.—Subclavian aneurysms are usually of slower course than axillary. Stationary aneurysms and aneurysms controlled for some time by a simple padded cap have perhaps been more frequent here than elsewhere, and spontaneous cure seems to have been more common in this than in any other situation. A few of these cures have been brought about by inflammation and even sloughing of the sac. Rupture may occur into the tissues; externally, in the pleura or bronchus.

The third part of the artery is by far the most frequently affected, the second very rarely except by extension from the third. An aneurysm of the third part usually varies from a walnut to an apple in size, and lies above the clavicle just outside the edge of the sternomastoid. It tends to pass down behind the clavicle, and thus to render certainty as to the exact seat of the aneurysm an impossibility (subclavio-axillary). Arterial hæmatomata and diffuse aneurysms push their way in diverse directions—up even to the jaw, down into the thorax and axilla, back under the scapula almost to the spine. The displacement of the arm and shoulder can be imagined, and dropping of the head towards the shoulder, to relieve tension, is natural. The subclavian vein is often compressed, sometimes leading to gangrene; so also the external jugular and its tributaries, leading to œdema and thickening of the side of the neck and above the clavicle, which may greatly complicate treatment. The brachial plexus may suffer even more severely than in axillary cases, the pain being often described by observers in the strongest superlatives. Paralysis may be complete. It is said that the phrenic may be irritated or compressed.

Diagnosis.—The common difficulties previously referred to are encountered. The occasional occurrence of a cervical rib over which the artery runs should be remembered.

Treatment.—Souchon has collected 115 cases—several being subclavio-axillary—treated by almost every known method. Many of the cases date from the septic era, and afford no certain ground for conclusions as to the value of the various operations practised. The whole question has still to be worked out with asepsis and sunk ligatures applied so as not to rupture any arterial tissue.

Cures by direct *compression*, by means of a soft rubber ball suitably fixed over the aneurysm, have been recorded; proximal compression has succeeded when the artery passed over a cervical rib on which it could be compressed;

Macewen regarded as an aneurysm of the first part of the left subclavian one in which needling yielded the best result. Here, where the dangers of operative treatment are so great, all reasonable non-operative means should be employed before passing to operative measures.

The results of these have hitherto been extremely bad; but, curiously enough, not nearly so bad upon the left side as upon the right. Wyeth suggests as an explanation of the difference that the vessels on the right are exposed to the direct rush of blood from the heart.

With regard to *arterial hæmatomata*—what should be done? In Lewtas' case ligature of the innominate and carotid succeeded—the wound lying beneath the scalene. In Twynam's the patient died of cerebral complication; post-mortem, an opening was found in the first part of the subclavian close to the origins of the vertebral, thyroid axis, and internal mammary. This does not seem satisfactory; yet in view of Lewtas' success, one cannot say that proximal ligature will not succeed. The alternative is to secure the vessel above and below the wound. This may be done in two ways: 1st, the method of Syme, *i.e.* introducing the finger into the hæmatoma through a hole which it plugs, feeling for the point at which blood escapes from the artery, and compressing this during the further operation. The opening being closed, the wound must be enlarged sufficiently to allow the vessel to be cleared proximally and distally to the wound and tied. Proximal control of the trunk would render this easy; but there is no case which shows whether a finger from such a wound can control the first part of the subclavian. 2nd, In a hæmatoma due to gunshot, Myles placed a temporary loop round the first part of the subclavian, and then having obtained control of the circulation, he opened the hæmatoma and tied the artery (3rd pt.) above and below the wound. The case recovered uninterruptedly. This necessitates two grave operations. Therefore it would seem that Syme's plan should be tried; if the ends cannot be secured during control by the finger a proximal loop may be added.

If the direct attack is put aside, nothing remains but ligature on the proximal side or plugging. Theoretically one would prefer to tie the second part to the first, and the first part to the innominate; but, according to existing statistics, ligature of the innominate and carotid comes out better than ligature of the first or second parts of the right subclavian.

Aneurysm of the Third Part.—If it is possible to tie the third or second part, this should be done. If there is no room, the question is an extremely grave one. H. Allingham has published a successful *ligature of the first part*, but recurrence necessitated excision.

Distal ligature of the third part has once been done with success, the carotid being also tied, as compression of it lessened pulsation in the sac.

Ligature of the innominate and carotid (as a guard against distal hæmorrhage) has been twice successful. Mitchell Banks also performed this operation successfully, but pulsation recurred the same evening, and was strong by the third day. The first part of the subclavian was then tied, and the patient died of hæmorrhage.

Excision of the sac has been done twice successfully, by Schopf (this was really a high axillary consolidated aneurysm pressing on the plexus and taken for a tumour) and by Halsted, and unsuccessfully by Moynihan. Both Halsted and Moynihan resected a piece of the clavicle; Halsted took it away with the aneurysm after he had tied the left subclavian "where it issued from the thorax." Moynihan sutured the piece of clavicle into place. His patient died from rupture of a small sac on the proximal side of the proximal ligature. The wound had healed, the patient was up, and the ends of the artery were well closed. Taking into consideration the freedom of the circulation in this region and the frequency with which pulsation recurs after the proximal ligature—than which no operation has been more deadly—primary excision seems to deserve a fair trial.

Amputation at the shoulder, with ligature of the axillary as high as possible, is the last operative method in these cases. The aneurysm may have consolidated in one case out of five.

Hæmatomata and Aneurysms on the Left Side.—For some reason unknown, ligature of the first and second parts of the subclavian has yielded far better results than on the right.

Vertebral Artery.—Hæmatomata from stiletto wounds have been pretty often recorded. They have sometimes been diagnosed as occipital because pressure below the sixth costal tubercle acts upon both carotid and vertebral; above this point the carotid only can be compressed.

Treat by incision and plugging with iodoform gauze, or by ligature after cutting away an anterior tubercle above and below.

ANEURYSMS OF THE NECK

EXTERNAL CAROTID.—Aneurysms of the external and internal carotid are rare. I have notes of a case apparently due to embolism, and of another attributed to a blow. Their nature was obvious. The sac of the first mentioned pressed on the hypoglossal and caused hemiatrophy of the tongue. Ligature of the common carotid was the treatment, until surgeons recognised the danger of embolism and softening after this operation. Of eleven cases only five were cured, while three died. Ligature of the external carotid was done only once and failed (Delbet). The obvious reason for these failures is the excessively free anastomosis. Excision is indicated. If there is good reason for not performing it, proximal ligature as near as possible to the sac should be done, and be followed by excision in case of failure. The common carotid should not be tied if there is room for a thread between it and the sac.

INTERNAL CAROTID.—Aneurysms may be intra-cranial (*q.v.*) or cervical. The latter are very rare. They cause a swelling which bulges into the pharynx, pushing the tonsil and soft palate before it. The circulation in the mucosa is impeded, and the parts look smooth, tense, and purple, closely resembling an abscess—for which it has more than once been opened. It may press upon any of the many nerves by which it is surrounded, and interfere with the cerebral circulation. A loop on the artery may simulate an aneurysm. Treatment by compression is not very successful, and proximal ligature of the common carotid has had a high mortality. It would be better practice to tie the root of the internal carotid when possible, and thus shut out the anastomotic circulation through the external.

COMMON CAROTID.—*Causes.*—Arterial hæmatomata are rare. Sometimes a twist or violent grip of the neck is regarded as the cause of true aneurysm, but usually no course is known. This aneurysm occurs often in young adults, and syphilis may sometimes be strongly suspected as the cause; it is decidedly the commonest external aneurysm among women.

Seats.—By preference the bifurcation; then the root in the right side; but aneurysm may occur anywhere except at the root on left side, no specimen of which is known.

Course and Symptoms.—Carotid aneurysms usually go steadily and even rapidly on to rupture—externally, into the trachea or œsophagus—unless death is caused earlier by asphyxia or by cerebral embolism. Stationary aneurysms are not very uncommon in this region, but spontaneous cure is rare.

As the tumour enlarges it extends up the neck, displacing and compressing surrounding structures. Its form is ovoid, its axis parallel to the sterno-mastoid. The larynx and trachea are pushed to the opposite side and flattened by degrees; the œsophagus suffers similarly, as also the recurrent laryngeal. Thus are produced dyspnœa and dysphagia, the former being usually the more severe. These structures may form part of the wall of the sac; as may all the muscles around. Cough and hoarseness are frequent. The jugular vein and its tributaries are flattened, and cease to carry blood; collateral veins show undue fulness. The effects of pressure on the nerves of the cervical and brachial plexuses, and on the sympathetic may be recognised. Attacks of vertigo, fainting, dimness of vision, hemiplegia, and other cerebral symptoms may occur spontaneously or on handling the sac. The bruit may be a constant annoyance.

Diagnosis.—The common difficulties met with must be solved upon the lines indicated at p. 222, where *Pulsating Goitre* is considered. *Blood cyst* of the thyroid yields first blood-stained fluid and then fresh blood when it is tapped; it moves with the larynx unless adherent. A loop low down on the artery has simulated aneurysm.

Treatment. — The condition of the opposite carotid pulse should be known. Non-operative treatment — especially rest, diet, and iodide of potassium—should be carefully tried; for any method inducing gradual cure will be more likely to avoid cerebral troubles than those which suddenly deprive the brain of its blood supply. If proximal compression can be applied—either towards the spine and muscles, or by seizing the carotid between the finger and thumb—it should be employed. Every care should be taken in using this method to avoid mechanical disturbance of clot in the sac. Needling may be used alone or with compression. Proximal ligature comes next. It is a serious measure. The mortality has been 25 per cent.

On tightening the ligature *dangerous syncope* may result. The patient may pass from anæsthesia into fatal *coma*. Opposite *hemiplegia* may supervene at once, in a few hours, or after days, weeks, or even months: it may be accompanied by blindness on the same side. *Convulsions* may ensue in the more acute cases, and death is the usual result. Minor signs of *disturbance of the cerebral circulation* are common. *Pulmonary* and *laryngeal symptoms* — dyspnœa, cough, pneumonia—have frequently caused death. They have been explained by injury to the vagus and its recurrent branch and by sepsis.

These grave complications have occurred in 20 per cent of the cases. Immediate results are due to cerebral anæmia, and imply imperfect supply through the other cerebral arteries. Syncope may pass off and leave no trace. Hemiplegia coming on after recovery from the immediate effects of the ligature may be explained by spreading thrombosis in cases in which blindness occurs—the ophthalmic artery being thrombosed before the origin of the cerebrals is reached. This thrombosis is doubtless favoured by a feeble collateral circulation. But in the majority of cases, hemiplegia is probably due to embolism of clot swept from the bifurcation of the carotid by blood running from the external into the internal carotid. Thrombosis, after aseptic ligature, is absent or very limited: it is thought that continued thrombosis, soft clot, and detachment of particles are connected with a mild, non-suppurative infection.

Suppuration of the sac has been frequent, usually from septic ligature; yet not always, for it may occur before treatment, or months after healing. Recurrent pulsation has been not infrequent after proximal ligature, though the artery has no branches.

Distal Ligature of the terminal branches, combined with proximal of the trunk, has been suggested to prevent this and embolism. Unless the sac were small and favourably situated, the operation would be difficult and the sac exposed to much disturbance. Distal ligature (Brasdor) alone is the treatment of aneurysm of the root of the vessel. The mortality has been heavy, but chiefly septic.

Excision of carotid aneurysms and hæmatomata has been practised, the reasons being recurrent pulsation, error in diagnosis, and impossibility of reaching trunk below except through the aneurysm. Good recovery followed in every instance. As in other cases, the question as to how often this method should be employed is now under consideration. Some hold that the risk of embolism would be reduced by removal of the sac, and this is likely enough if the roots of the internal and external carotids are tied.

Arterio-venous communications, almost invariably direct and due to stabs, have formed between each of the carotids and the neighbouring great veins. The symptoms are usual, with the addition of constant annoyance and disturbed sleep from the bruit, and of various symptoms due to disturbance of the cerebral circulation.

If treatment is required, it can only be ligature above and below the wound in the artery.

Aneurysm of the Innominate.—*Causes.*—These aneurysms are among the more frequent of surgical aneurysms. They are among the largest known. No arterial hæmatomata or arterio-venous communications are recorded. Stationary aneurysms are rare; spontaneous cure still more so.

Seats.—The aneurysms may extend on to a dilated aorta, subclavian or carotid.

Symptoms.—Pain about the right sterno-clavicular joint, undue dulness behind the manubrium and first space, excessive pulsation in the presternal fossa, and perhaps a deep swelling here; a systolic murmur and a diastolic shock, the murmur being conducted along the subclavian and carotid; then come effects of pressure on the recurrent, vagus, sympathetic, brachial plexus; on one or both innominates or superior cava; diminution and delay of radial and temporal pulses; dyspnœa and dysphagia with obvious displacement of the trachea and œsophagus; displacement of the heart down and out without hypertrophy; protrusion of all bones around the sterno-clavicular joint, erosion of them, and free bulging beneath the skin. This forward course is common, but a tumour appears first just mesial to the sterno-mastoid. Death may be due to rupture externally, into the pleura, lung, trachea, œsophagus, or to pressure on one or other of these canals. Hypostatic congestion and bronchopneumonia occur from pressure on the vagus or tracheal obstruction.

Diagnosis of "Aneurysms at the Root of the Neck."—Such aneurysms may spring from the root of the carotid, the first part of the subclavian, the innominate, or the arch of the aorta: or may involve several of these parts. It is often impossible to make an exact diagnosis. A skiagram and the screen may conceivably help. The points to which chief attention must be paid are:—

The point of first appearance of the tumour. A carotid aneurysm appears between the heads of the sterno-mastoid, tending forwards beneath the inner head; a subclavian beneath and external to the clavicular head; an innominate in the presternal fossa, tending up and out behind the sterno-clavicular joint; an aortic sac may occupy any one of these positions.

Form.—All are either globular or ovoid. If they have a long diameter it is vertical in the carotid, nearly transverse in the subclavian, variable in the innominate, which may extend along either the carotid or subclavian, and usually vertical in aortic. The aortic and innominate arise within the thorax, so the fingers get no sensation of a sac narrowing below: in the carotid and possibly in the subclavian this idea may be gained.

Evidence of a large tumour in the mediastinum, *e.g.* pulsation and dulness in the second and third right spaces, is much in favour of aortic as opposed to innominate.

Bruit is usually conducted best along the artery involved; but in the innominate it may be conducted unequally along its two branches. It is therefore of little value.

Pulse.—The pulse wave reaches the peripheral branches of the artery, in the root of which an aneurysm is situate, later than on the sound side. If not detected by the finger, this retardation may be shown by tracings simultaneously recorded. The tracing on the affected side should show the peculiarities of the aneurysmal pulse. Lessened volume and even loss of pulse are not characteristic of aneurysm.

Even on paper various states of affairs may be arranged with the aid of an aortic sac placed at one spot or another, of which it would be difficult to foretell the exact result upon the pulse. But an aneurysm of the ascending aorta should delay all the pulses equally, *i.e.* have no detectable effect upon the two radials. An innominate aneurysm should delay and give aneurysmal characters to both right radial and temporal pulses. An aneurysm of the aortic arch should delay the left radial pulse, and so forth.

Pressure Signs.—These must be carefully considered, and a conclusion drawn as to which aneurysm is most likely to produce them all.

Tracheal-tug.—This indicates an aortic aneurysm.

Treatment.—Non-operative treatment should be tried. Then needling, coupled with compression of the common carotid. This failing, simultaneous ligature of the right carotid and subclavian arteries should be done, unless one is already impervious. Of late years this ligature has met with a large measure of success. The subclavian has almost always been tied in its third part.

ANEURYSMS OF THE LOWER LIMB

Below the bifurcation of the popliteal aneurysms are rare, even including arterial hæmatomata and aneurysms in the production of which injury takes a very large share; and such constitute the large majority here.

DORSAL ARTERY.—Edmunds could find only six apparently spontaneous aneurysms.

THE PLANTAR ARTERIES are very rarely affected, and only as the result of injury. These aneurysms and hæmatomata should be excised.

THE TIBIAL AND PERONEAL ARTERIES may be wounded, torn by contusions or lacerated in fractures. A few spontaneous aneurysms, usually following some strain, are recorded; and a few seem to have been due to embolism.

Hæmatoma from wound is most frequent in the lower third; from fracture in the upper and middle thirds. Here, as elsewhere, the immediate diffuse hæmatoma is much more dangerous than the circumscribed, which forms slowly but may ultimately threaten the life of the part. Both, fortunately, are rare. They closely simulate deep abscess.

Aneurysms arising deep in the front or back of the leg, cause at first only doubtful symptoms — pain, cramp, impaired function — with deep indefinite swelling. In such a case, however, Delbet heard a murmur distinctly and found later a nut-sized aneurysm of the posterior tibial. As the aneurysm increases and diffuses, symptoms of pressure on veins and nerves become marked, and more or less extensive gangrene may occur. So, too, may suppuration. These aneurysms are difficult to recognise, difficult to locate exactly, and difficult to treat.

In the matter of simple recognition the bruit is important. In endeavouring to refer the aneurysm to its artery, the pulse, the conduction of the bruit, and the level of the point of greatest swelling and pulsation must be relied upon. The greatest difficulty arises in aneurysms of the end of the popliteal and commencement of the tibials. The post. tibial may be felt pulsating over a sac on the anterior tibial.

Without a good history the diagnosis of suppuration may be impossible. The state of the pulse below is very important.

Treatment.—Easily accessible hæmatomata and aneurysms should be excised. Diffuse hæmatomata must be incised and the vessel tied. If a fracture is the cause, the bones should be sutured. In bad fractures thus complicated the question of amputation will arise. Circumscribed hæmatomata may get well while the fracture is in splints; naturally, the toes must be carefully watched.

In deep circumscribed hæmatomata and aneurysms, elevation, compression of the femoral, or Reid's bandage may be tried. These failing, the popliteal may be tied. But if there is much circulatory difficulty, the sac should be removed. The greatest difficulty would arise in dealing with a sac on the anterior tibial between the bones.

Popliteal Aneurysm.—These constitute more than half of all cases of external aneurysm (128 of 240 cases). Hæmatomata are rare, but may arise from stabs or fragments in supra-condylar fractures. A few aneurysms have seemed due to friction against exostoses and abnormal spines of bone; a few to embolism. The great majority are spontaneous and occur in men. Symmetrical aneurysms are not uncommon. A popliteal aneurysm has very rarely opened into its vein; but a good many cases of direct communication from wound have been recorded. The special frequency with which this vessel is affected has not been certainly explained. Some point to the effect of full flexion in kinking it, intercepting the flow of blood along it and suddenly raising the pressure, but this movement is not very common; others think that it is the longitudinal strain due to extension and over-extension which does the mischief; some blame both movements. Broca thought that the arch of the Soleus compressed the artery when the muscle contracted; but, being fibrous, it can hardly do so, and contraction of the muscle would rather draw it from the artery. A special relation to various callings (sailors, lamplighters, jockeys, postilions, carriage-footmen, tailors), often said to exist, rests upon slender evidence. It occurs in young people, the average age being about thirty-five, before the age of marked atheroma; and it has never been shown that the popliteal is, like the aortic arch, a point at which atheroma is specially liable to appear. A history of syphilis is not disproportionately frequent. Bits of the artery excised with aneurysms are described as healthy. Its special frequency among the labouring classes seems certain, and its apparent origin during effort is common; but what the condition is which causes the popliteal artery to yield rather than another is not clear.

Seat.—The sac may spring from any part or aspect of the vessel, displacing the trunk (when possible) in the opposite direction. This displacement is most marked when the sac intervenes between the artery and the bones; more commonly it lies on the dorsal aspect of the vessel. Widely as the sac may cover and even adhere to the artery, the mouth is usually of quite moderate size.

A femoro-popliteal aneurysm, protruding through the adductor opening, is rare; a popliteo-tibial, passing under the arch of the soleus, very rare. These aneurysms fall naturally into a high and a low group. The high, occupying the large space above the condyles, do not cause such early or such urgent symptoms as do the low, which are compressed between prominent bones, dense fascia, and a powerful muscle. Further, the low group require for their cure obliteration of numerous articular and sural arteries—difficult to bring about, and when effected offering considerable opposition to the establishment of the collateral circulation. The low group, therefore, closely resemble the rarer, but important, group of high tibial aneurysms.

Symptoms.—Often pain or sense of something giving during an effort is noted. Occasionally weakness of the knee or leg or œdema of the foot first attracts attention. Next come difficulty in using the knee, pain in the bone and down the leg, attributed to rheumatism, and a pulsating swelling.

Nowhere are the symptoms of aneurysm more typically seen. If untreated the sac enlarges, as a rule, pretty quickly; it protrudes backwards and more often inwards than outwards. Extension and full flexion of the knee become impossible; it is kept constantly more or less flexed and extends slightly with each systole. Flexion is especially marked in low aneurysms on which pressure is greatest. Swelling supervenes; pain becomes more and more severe, usually along the tibial nerve; paralysis is unusual. In anterior aneurysms the boring pain of erosion of bone may be marked. Patches of gangrene may appear or more extensive portions of the limb may die. More commonly before this occurs the sac ruptures—into the connective tissue, through the skin, or into the knee joint. The latter is a rare complication, and is characterised by pain and sudden distension of the joint by fluid which often pulsates and is more or less reducible into the sac when the femoral artery is controlled.

It is not very uncommon to find a moderate hydrarthrosis when the sac presses on and adheres closely to the posterior ligament; this does not pulsate.

Suppuration of the sac is rare.

Diagnosis.—This is usually simple. The ordinary difficulties may be met with (p. 222) from bursal cysts, sarcomata or simple tumours lying deep to or on the artery.

Treatment.—It was usual to begin with compression of one kind or another. Now, ligature is usually performed as the method of choice, either in Scarpa's space or Hunter's canal. The result of this, calculated from seventy-six modern hospital cases is:—sixty-four cures of the aneurysm (88·1 per cent); but of these seven (9·2 per cent) recovered after gangrene; whilst another—in very feeble health at the start—had gangrene and died. In eight pulsation recurred. In other words, 10·5 per cent suffered from gangrene and one died; and in another 10·5 per cent the operation failed to cure. It is probable that ligature of the popliteal will be more used in the future and with better results both as regards gangrene and failure. Perhaps excision or incision of the sac will prove the best treatment of all; but it has not yet been fully tried. This operation may save a limb after rupture or after suppuration, when ligature might only add to the gravity of the case.

Aneurysm of the Femoral Artery.—These are three to four times less common than popliteal aneurysms, to which they rank next in point of frequency. Women rarely suffer. They are more frequent above than beneath the sartorius and are usually spontaneous. A good many *arterio-venous communications* — direct and indirect — from wound have been described.

Symptoms.—These are very clear and usual. In form the sacs above the sartorius are usually rounded, beneath it flattened. The surroundings not being unyielding the vein is not heavily pressed on as a rule; and the nerves chiefly liable to pressure are the long saphenous and internal cutaneous. The course of these aneurysms is onward, but often somewhat slowly. Stationary aneurysms and spontaneous cure may occur, but are rare, especially the latter.

Diagnosis.—Is simple as a rule. Difficulty may arise from the rare occurrence of an aneurysm upon a branch in the line of the artery. At St. Bartholomew's (Rep. '97) a young man *with double aortic* disease had a deep, ill-defined, hot, tender swelling in Scarpa's space *over which the femoral pulsated;* it was incised and clot turned out, when an aneurysm as large as a walnut in the profunda was found. Israel cut into another doubtful swelling and found it to be an aneurysm of the third perforating. No aneurysm off the main line will affect the tibial pulse. This is the trunk upon which multiple aneurysms (2-4) have been most often observed. Symmetrical aneurysms may occur.

Treatment.—Proximal digital compression may be tried if there is room without injuring the sac; it has yielded good results. Macewen's first success with needling was in this situation. If ligature is chosen it should be applied close above the aneurysm, wherever it may be. Should it fail, as it not uncommonly has, excision can be proceeded with. This should be done at once in diffuse aneurysm. The operation is at its easiest in this region; and the results have been quite satisfactory.

INGUINAL ANEURYSM.—Ilio-femoral aneurysms, developed above and below Poupart's ligament, are necessarily taken together. They are often excited by strain or some more direct injury. This is the commonest seat (outside the lungs) of aneurysm by erosion from spread of inflammation (chiefly from buboes) to the coats of the artery; but such cases are rare.

Symptoms.—A bilobed swelling divided by Poupart sometimes more above, again more below it—having all the characters of aneurysm—enlarging rapidly in the iliac fossa, less rapidly beneath the fascia of the thigh, compressing the femoral vein early, and therefore causing œdema and tending specially towards gangrene; irritating the genito-crural and perhaps other nerves, and causing much pain by tension behind Poupart. It tends to rupture externally, or into the iliac fossa. Erosion of pelvic bones and rupture into the hip-joint have been recorded. Cure by sloughing has also occurred.

Diagnosis.—Is often simple, but may be difficult when it is chiefly iliac. Pulsating sarcoma of the hip-bone resembles it when typical. When a fluid aneurysm ceases to pulsate, it may be taken for a chronic abscess; when it becomes inflamed, for an acute abscess.

Treatment.—Compression has been tried by lever on the common iliac and by the aortic tourniquet—often coupled with an elastic bandage up to the aneurysm or some other form of distal compression. Of seven cases the results were two cures, one early recurrence, three failures, and one ruptured sac. Sixteen cases of proximal compression yielded three cures without accident (Delbet). Transperitoneal ligature of the external iliac has not been done many times, but has yielded favourable results so far. When not forced to go so high Cooper's method may be used. The great danger after either of these ligatures for a large, possibly diffuse aneurysm, is gangrene. This occurred thrice in sixteen recent cases (Delbet) with two deaths after amputation; and one, if not two, other cases narrowly escaped gangrene. Such results naturally lead once more to the question of excision, which Delbet advocates even here. There are really no statistics to go upon. Unless the circumstances are very favourable, operation on an aneurysm in the iliac fossa will be difficult, and may necessitate the division of Poupart. Control of the circulation by finger above the aneurysm should, of course, be obtained; the epigastric might be tied early in the abdominal wall. The objection that the profunda must often be tied is of little weight, for it is tied only when opening into the sac, and cure cannot occur after ligature, etc., unless it is closed. The results of excision, however, have still to be worked out.

GLUTEAL AND SCIATIC ANEURYSMS are usually connected with wounds, contusions, or fractures of the pelvis; many are arterial hæmatomata. Of thirty cases, twenty-one were gluteal, nine sciatic. They occur chiefly in men.

Symptoms.—These develop rapidly in cases of diffuse hæmatomata, more slowly in circumscribed hæmatomata and aneurysms. In the latter, pain and other evidence of pressure on nerves first occur—then swelling, deep, fixed, poorly outlined, pulsating. The other signs of aneurysm are present. It enlarges steadily or by leaps; sometimes remaining long stationary, possibly undergoing consolidation; but usually passing on to rupture into the connective tissue or externally.

Diagnosis.—The first thing is to make out whether there is any intrapelvic portion. Next to eliminate pulsating sarcoma. Lastly to endeavour to decide by the situation or the history of the point of appearance of the swelling whether it is gluteal or sciatic.

Treatment.—This lies between transperitoneal ligature of the internal iliac and excision of the sac. The latter has been done several times with good results; but it is difficult undoubtedly. As a rule it has been done without controlling the circulation; but a finger could be safely placed on the internal iliac. A great difficulty would arise if it were discovered at the operation that a portion of a gluteal sac was intrapelvic; if the artery could be clipped, the forceps might be left on. But ligature of the internal iliac would be the safer treatment.

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Angina Ludovici. *See* NECK, SUPPURATION.

Angina Pectoris.

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SYNONYMS—*Syncope Anginosa; Angor Pectoris; Suffocative Breast-pang.*

DEFINITION.—A spasmodic attack of pain or oppression in the region of the heart, often radiating down one or both arms, and accompanied even in its slighter forms by the necessity for ceasing exertion, and in its severer forms by a feeling of impending death.

SYMPTOMS.—The most descriptive name which has been given to angina pectoris is that of "suffocative breast-pang," for the word "pang" indicates the spasmodic and intense nature of the pain which characterises the disease, the word "breast" indicates its position, and the word "suffocative" indicates the peculiar sense of breathlessness by which the pain is accompanied.

A good deal of discussion has taken place regarding the nature of the pain and oppression felt in angina pectoris. It is probable that this is owing in great measure to a confusion between two things easily distinguished by those who feel them, but very hard to describe to others.

Almost the only way to understand these feelings is to make a very simple experiment upon oneself. On pressing a pocket-handkerchief over the nose until the desire for breath becomes overpowering, a distinct discomfort in the chest and feeling of suffocation is felt, but it is of a different nature from one which can be produced by another experiment. This consists in pressing the thumb or finger backwards and inwards over the carotid, so as to compress it against the cervical vertebræ. In this way not merely the carotid but the vagus, and probably also the sympathetic nerve are compressed and a feeling of suffocation occurs quite as unbearable as that which is produced by holding the handkerchief before the nose but of a very different character. This feeling is one of intense oppression over the chest which seems to drive the chest in, and at the same time prevents one from drawing a breath, although the air-passages are perfectly open, and there is no reason whatever why one should not take a deep breath. This feeling resembles closely the feeling of oppression caused by very violent exertion, with this difference, that after exertion there is a great desire to draw frequent and deep breaths, while compression of the vagus does not produce this effect. It is, I think, the difference between the two sensations of suffocation from closure of the nostrils and compression of the vagus that has led to a great deal of dispute about the feelings of the patient in angina pectoris.

It is to be remembered that angina pectoris, like toothache, is a symptom rather than a disease. Toothache is usually caused by a cavity in a carious tooth, but pain may be felt in the teeth during an oro-nasal catarrh, although the teeth are perfectly sound. In like manner angina pectoris is usually due to atheroma of the coronary arteries of the heart, but pain very much like that of angina may occur from other causes.

Just like toothache, angina pectoris varies very much in degree, from a slight pain, which is hardly to be observed, to the most intense agony. In nearly all cases, even when it is slight, it appears to be accompanied by a feeling of oppression. The difference between the pain alone and the pain accompanied by oppression is best understood by considering that even in very slight cases of angina the patient on walking up a hill is almost always obliged to stop. Yet the patient will confess that the pain in the chest has not been so severe as the pain of a twisted ankle or even the pain of a bad corn.

And yet in spite of a twisted ankle or a bad corn a man may walk uphill without showing almost any sign of wishing to stop, and therefore I think that it must be the oppression rather than the pain that makes the man stop and make him feel that he cannot go on. In more severe cases the pain not only comes on during exertion, but often does so without any apparent cause, and it also becomes much more severe. In commencing cases the pain is slight, its duration is short, and recovery is rapid. After resting for a minute or two the patient feels better and may sometimes be able, without much discomfort, to resume the walk. Moreover, if the pain is so slight that he is able to persevere in spite of it, the pain will generally pass off, and the patient will be able to go on walking afterwards without discomfort. But when the pain is severe the patient is obliged to stop still, and sometimes is unable to make any movement whatever. If he is standing when the pain comes on, he will try and lay hold of something, and rest against it so as to take off any weight upon his chest. If he is lying in bed, the chances are that he will raise himself to a sitting posture, and then lean his hands against his knees so as to support himself in that posture, which is so frequently observed in cases of aneurysm. In such severe cases one generally finds that the face has an anxious, distressed look; sometimes the look of distress is exceedingly great. The face, as a rule, is pale, sometimes the lips may have a slightly livid tinge, but the paleness is more marked than the lividity. The cheeks look somewhat sunken, and the surface of the skin is very often cold and covered with sweat. The respiration is usually hurried and very short. It occurs in little quick gasps, the patient feeling that he cannot expand his chest, and yet if he is told to take a deep breath he will probably take one with perfect ease and feel all the better for it.

Duration.—These symptoms sometimes come on very suddenly, last only a short time, and pass away suddenly. In other cases they come on suddenly, last over a period varying from several minutes to two or three hours, and then they may pass away either suddenly or gradually.

Nature of the Pain.—This is variously described by patients. Some compare it to a severe pressure, like a heavy weight or like an iron hand compressing the chest. Others complain of a feeling of distension from within as if the heart would burst. Others describe it as of a stabbing character.

Site.—The site of the pain is usually over the sternum, sometimes at its lower and sometimes at its upper part. At other times it is more to the left, nearer to the apex of the heart. Occasionally the pain as it passes off may leave the left part of the chest, while it still remains to the right side of the sternum.

Radiation.—In severe cases it almost invariably radiates to some extent, generally down the left arm, sometimes down the right, sometimes down both, and sometimes to the shoulders and up the neck as well.

Pulse.—The pulse during this condition is almost always small. Some observers have stated that it is unaltered in strength or rhythm, but this, although it may possibly occur sometimes, is not a usual condition. A sphygmographic tracing shows that the pulse is very small, and on feeling it one may observe that the pulse wave is very slight. Occasionally the pulse is slow, but generally it is quick, not unfrequently it is hard and somewhat difficult to compress.

ETIOLOGY.—Amongst the predisposing causes of angina pectoris may be mentioned age and sex. True angina occurs more commonly in men than in women, and almost always after middle life. In perfectly young people, however, symptoms very nearly simulating those of true angina may occur. Just as in a perfectly healthy girl there may be the symptoms of paralysis, so there may be in a healthy girl the symptoms of angina pectoris. Angina pectoris is conveniently divided into two classes, true angina or false, or pseudo-angina. The true is a very serious, often fatal disease; the false is generally recovered from completely. True angina is a disease which chiefly affects men at or above middle age. Pseudo-angina chiefly affects young women.

Other predisposing causes of angina are syphilis, gout, and rheumatism. Syphilis leads to endarteritis, and so tends to render the arteries thicker, more friable, and more liable to become blocked. Gout has a somewhat similar action, and tends, moreover, to affect the kidneys and give rise to a high arterial pressure. Rheumatism affects the valves of the heart, and by the changes that it induces there it tends to block the coronary arteries.

Distension of the stomach, either by food or flatulence, increases the tendency to angina on exertion, and may indeed bring on an attack apart from any exertion. The passage of flatus either upwards or downwards usually gives some relief, and sometimes very great relief.

Social Position.—It is said by some to be rare in hospital patients as compared with those in better circumstances. It is certainly far from uncommon amongst hospital patients, but I think that angina uncomplicated by any gross lesion of the heart or vessels is more common amongst the wealthy than the poor. This is what one would naturally expect, for atheroma naturally leads to gross lesions in persons who are subjected by their daily work to severe muscular exertion, and consequent strain of the heart and vessels.

Amongst the wealthier classes, men of literary pursuits are said to be especially liable to it, and this too is natural, for such pursuits are usually connected with sedentary habits, and lack of exercise, sometimes combined with free-living, naturally tends to cause degeneration of the heart and vessels.

Emotions.—Worry and grief are certainly predisposing as well as exciting causes.

Diseases. — Infective diseases, such as typhoid fever, erysipelas, and pneumonia, predispose to angina by weakening the heart, but the worst of all is certainly influenza, which appears to me to have a specific power of attacking the heart, and weakening it as much in four or five days as typhoid fever will do in three weeks.

Certain poisons, and especially alcohol, tea, and tobacco, predispose to angina. This is what one might *à priori* predict in regard to the latter two substances, for theine or caffeine, which is the active principle of tea, and nicotine, which is the active principle of tobacco, both act on the heart and vessels, stimulating them to increased contraction, and thus tending to cause a rise in the blood pressure, such as forms one of the most important factors in the production of angina.

Exciting causes.—The exciting cause of an attack is generally some call upon the heart for extra exertion, which is made either by some unwonted effort or by some unwonted emotion. The effort which most frequently brings on the attack is that of walking uphill, and more especially when this is attempted shortly after a meal. Distension of the stomach or intestines by food or flatulence may also bring on an attack even without exertion. Violent emotions tend to bring it on also, but all emotions do not tend to bring it on in the same degree. The softer emotions, pleasure, affection, love, and so on, are less liable to bring on an attack, unless they should be very sudden and very excessive. Sometimes great joy affecting a person suddenly and unexpectedly has caused sudden death, but joy, as a rule, does not kill, whereas anger very frequently does. John Hunter suffered from angina pectoris for a number of years before his death, and was accustomed to say that his life was at the mercy of any rascal who cared to irritate him. The end of his life was that one day at a Committee Meeting he was suddenly contradicted; he left the room, staggered into an adjoining apartment, and fell down dead.

PATHOLOGY.—The heart was found by Harvey not to be sensitive when touched, but the stomach and intestines are only slightly sensitive to touch externally, and yet are liable to acute pain from distension or spasmodic contraction. The same thing is observed in the arteries in migraine, which affords the closest analogy with angina pectoris. The exact cause of the pain in angina pectoris cannot at present be stated with certainty, but the condition under which it comes on is a disturbance of the normal relation between the power of the heart and the resistance it has to overcome, just as in migraine there is a disturbance of the normal relation between the proximal and peripheral parts of the same artery.

Thus a strong heart readily continues to beat in spite of the resistance in the arteries being increased by exertion, by exposure to cold, by reflex vaso-motor excitement, or by such conditions of the blood as occur in gouty kidney. A feeble or fatty heart also may continue to beat steadily, so long as the pressure it has to overcome in the arteries does not exceed its powers.

But if the relationship is disturbed by opposing a great resistance to a weak heart, pain is apt to come on. More especially is this the case if the cardiac weakness be due to imperfect supply of blood, and the lesion above all others which is associated with angina pectoris is narrowing of the lumen of the coronary arteries. It would appear that the reason why this lesion is associated with angina is that when the heart is called upon to make any extra exertion it requires, like other muscles, an extra supply of blood. When the coronary arteries have their lumen lessened this extra supply of blood is not forthcoming, and then some affection of the muscle or nerves of the heart occurs, which leads to the symptoms which we know as angina. This narrowing of the lumen may occur either in the coronary arteries supplying the right or left ventricle, but it is chiefly when the arteries supplying the left ventricle are affected that symptoms of angina occur. If the arteries supplying the right ventricle are affected, a different class of symptoms is produced, to which the name of "cardiac asthma" has been given. The patient is short of breath on the very least exertion, while his lungs are perfectly healthy, but he does not as a rule tend to suffer from severe pain.

VARIETIES.—Angina pectoris is usually divided into true angina and false or pseudo-angina. This division is objected to by some authorities; but, as Osler very truly says, its clinical convenience outweighs any disadvantages it may have. The difference between these classes of angina is not one of degree, it is a difference of kind. The pain and distress in an attack of pseudo-angina may be very much greater than in a slight attack of true angina, but the prognosis is very different in the two cases, for there is little or no risk to life in the apparently severe attack of pseudo-angina, while there is cause for anxiety from even the slightest attack of true angina.

The main difference between true and false angina pectoris is that true angina such as occurs in men beyond middle life, is a disease characterised by more or less definite lesions of the heart or vessels, having a course which almost invariably tends towards death. Pseudo-angina, on the other hand, whilst it may present symptoms apparently almost identical with those of the true disease, is usually observed in young girls, or in men before middle life, who indulge to excess in tobacco or tea. It has no definite physical basis, and may be looked upon as a functional disease of a hysterical or neuralgic nature. Moreover, the treatment which cures or relieves the true disease is of little use in these cases of hysterical angina.

Vaso-motor Angina.—In this form, which was first described by Nothnagel, the coldness, pallor, and sweating which generally occur in severe cases of angina are even more marked than usual, and there is a tendency to lividity of the extremities. In ordinary angina the attacks usually occur from the pressure in the arteries being raised by exertion; but in this form the contraction of the vessels and consequent rise of pressure occur from the application of cold such as by washing the hands in cold water. The spasm of the vessels in this condition resembles that in Raynaud's disease. Such vaso-motor spasms may occur at various ages. They are closely allied to the arterial spasm which occurs in migraine, and like migraine are more common before than after middle age. As the heart is not likely to be weakened by atheroma or fatty degeneration before middle life, vaso-motor angina is not as a rule dangerous to life unless the patient is elderly or unless the heart has undergone premature degeneration.

Like migraine, this form of angina may pass off with advancing years, and the patient may recover completely from the attacks. On the other hand, it may occur along with degeneration of the coronary arteries and heart, and then its consequences will be the same as if the blood pressure had been raised in the usual way.

Vaso-motor angina may thus belong either to the class of true or pseudo-angina, according to the conditions of the heart and vessels in which it occurs.

Syphilitic Angina.—I have seen two cases in which the symptoms of angina coincided with the growth of syphilitic nodes in the sternum, and disappeared along with the nodes themselves during the administration of iodide of potassium. It is difficult to say whether the attacks in these cases were of a reflex nature due to the irritation of the node, or whether they were caused by some other condition which was removed by the iodide along with the node.

Reflex Angina. — As I have already mentioned, anginal pains may occur when the blood pressure to be overcome becomes excessive for the heart. Irritation of sensory nerves tends to cause contraction of the blood vessels, and thus such conditions as cause severe pain tend to bring on anginal attacks if the heart be already weak. This is, I think, the explanation of some cases of anginal pain occurring from painful distension of the stomach or colon by flatus, or from the passage of a calculus down the bile duct or ureter.

At the same time it is to be remembered that distension of the stomach by flatus or food may cause a certain amount of displacement of the heart, and thus interfere mechanically with its action.

Toxic Angina.—The excessive use of tobacco, tea, or coffee may cause angina which is often of the vaso-motor type. It is more common in patients under middle age because the fright which such attacks give to the patients leads them to use the articles in question with great moderation, or to give them up entirely before any senile or degenerative changes have occurred in the heart. It is therefore as a rule recovered from, but, like vaso-motor angina, it may be very serious if the heart or coronary arteries have undergone degeneration either from advancing years or other causes.

DIAGNOSIS.—The symptoms of angina may be simulated by hysteria and by calculus in the bile duct. In hysteria the sense of suffocation associated with severe pain in the cardiac region might lead one at first sight to say that the patient was suffering from angina pectoris, but such patients generally point to the throat as the place where the difficulty of breathing exists, and these cases are observed in young women. On examination of the heart, it will probably be found to be perfectly healthy, and even should a valvular murmur exist the probability is that the case is not one of true angina. Moreover, there are usually other symptoms of hysteria, such as emotional displays, which may alternate with complaints of cardiac pain, and copious discharge of pale urine after the attack is over.

The symptoms of angina—the sudden accession of the pain, the apparent difficulty of breathing, the paleness, the cold sweats, may be simulated by the passage of a calculus down the ureter or of a calculus through the bile duct. The difference is that in the passage of a renal calculus the pain, as a rule, radiates downwards towards the bladder or towards the genitals, and the pain of a calculus passing through the gall duct tends to radiate more or less laterally, sometimes a little downwards towards the umbilicus, sometimes even a little upwards towards the shoulder, but generally towards the right shoulder and not down the arm. There are some cases of biliary calculi which are very difficult to diagnose from angina pectoris, because in cases where a calculus in the bile duct and a weak heart coincide, the irritation of the calculus passing through the bile duct may give rise to some symptoms of cardiac failure which may be attributed to angina.

Toxic angina is best diagnosed by the fact that it ceases when tobacco, tea, and coffee are left off entirely. True angina usually occurs in men, more rarely in women above middle age. Even the first attack may be severe or fatal, but very often the history is that it has come on as a slight pain or feeling of oppression on trying to go uphill, running to catch a train, or other exertion, more especially soon after a meal. On careful auscultation a slight systolic murmur indicative of atheroma of the aorta may often be heard over the second right costal cartilage or first interspace close to the sternum. Sometimes this murmur is heard still more distinctly over the sternal end of the first rib. It might be very much localised, or may be conducted up towards the subclavian or carotids more especially to the right. The fact that pain or oppression comes on during exertion in a patient above middle age, and especially if the murmur just mentioned can be heard, renders the diagnosis of true angina most probably correct.

PROGNOSIS.—In hysterical angina the prognosis is good, and indeed the prognosis may be said to be good in most cases of cardiac pain in people below middle age, and where no fatty degeneration or other disease of the heart is present, and no indication is to be found of degeneration of the arteries. In persons above forty-five the prognosis must always be guarded, because the life of such persons is very uncertain. On the one hand, with care and attention, they may live for a good many years after the first attack of angina, but it is impossible to say when another attack may come on, and whether it may prove fatal or not. On this account patients suffering from angina, however slightly, ought to make provision for the possible occurrence of sudden death by putting their affairs in order and carrying with them a card bearing their name and address.

If patients are inclined to be careless it may be necessary to warn them very plainly of the risks they run, but if they are ready to take care of themselves, it is advisable to look upon their complaint in a hopeful way, and to inform them that with care they may possibly go on for years. This is all the more necessary as the depression of spirits, anxiety, and possibly even fear, which may be brought on by an unfavourable prognosis, tend to depress the action of the heart, and may bring about a fatal result.

TREATMENT.—In cases of hysterical angina the administration of nitrite of amyl, so useful in true angina, is of little or no use. In 1867, a few weeks after I discovered its power of relieving true angina, I tried it in a case of hysterical angina, but it gave no relief, although I pushed it so far as almost to produce asphyxia. More relief is afforded by hot applications to the chest, such as an india-rubber bag filled with hot water, or by carminative mixtures, such as the following:—

℞ Tinct. valerian ammon. Spt. ammon. aromat	āā ʒss.
Spt. chloroform	♏ x.
Tinct. lavand. co.	♏ xx.
Spt. vin. rect.	ad. ʒij.

M. ft. hst.

S.—To be taken in a wine-glassful of hot water, when the spasm is severe.

During the intervals inquiries should be carefully made regarding any source of mental disquietude, and it should be removed if possible.

The patient's attention should be directed as much as possible away from herself and engaged in working for others. Food should be plain and simple, but moderate exercise and plenty fresh air should be enjoined.

In cases of toxic angina complete abstinence from the toxic agent, tobacco, tea, or alcohol, must be insisted upon, as even a small quantity may keep up the condition.

In cases of true angina one of the first requisites in the treatment is first to insist strongly upon the necessity of avoiding any sudden exertion on the part of the patient. I have already mentioned that if a patient is able to continue exercise in spite of pain, the pain passes off after a time, and the exercise may be proceeded with without discomfort. Whatever the patient does should be commenced very gently. When he begins to walk he should do so very slowly indeed, and gradually quicken his pace as he goes on. He should not attempt anything likely to strain him, such as lifting or strapping a portmanteau and such-like exertion, and, above all, he should never hurry to catch a train or omnibus. These rules apply at all times of the day, but they are even more necessary after a meal, and it is advisable to let the patient lie quiet half an hour after a meal, before undertaking any exertion at all. If exercise be sufficiently gentle not to bring on the pain it is of advantage to the patient, because it tends to maintain the strength of the heart, and to increase the nutrition of the body generally as well as the arteries in particular. During an attack the best way of cutting short the difficulty of breathing and the pain is to let the patient inhale some nitrite of amyl. This may be done either from a bottle containing the drug in bulk, or from a glass capsule containing five minims, which is broken when required. Hot applications should also be put to the hands and to the feet. Nitro-glycerine or, as it is more conveniently called, trinitrin, is more convenient than nitrite of amyl and acts almost as well. The patient should keep in his waistcoat pocket a small box or bottle containing the tablets, and as soon as the pain comes on should begin to nibble one. If the pain be acute he should break it up quickly in his mouth and swallow the whole. If the pain is not very acute, he may take only a part of one, instead of a whole tablet. Nitro-erythrol is another drug which has a somewhat similar but more permanent action, so that half a grain of it may be given three times a day with great advantage as a prophylactic. This substance, whose full name is tetra-nitro-erythrol or tetra-nitro-erythrine, is sometimes called for shortness tetranitrin. It is most readily given in the form of tablets made up with chocolate in the same way as tablets of trinitrin. Each tablet usually contains half a grain of tetranitrin. In some cases where the pain is very excessive, and where it does not yield to the nitrites, a hypodermic injection of a third or half a grain of morphia may be given.

The drug which of all others is the best for removing the cause of the attacks of angina pectoris is iodide of potassium, and this may be given in doses varying from five to fifteen grains three times a day. A good plan is to give it for ten days or a fortnight at a time, leave it off and then begin again. When given intermittingly in this way it may be continued for months, or even years. In gouty patients the tension may be lowered by the administration of twenty grains of nitrate of potash along with thirty of bicarbonate of potash or soda in a large soda-water-tumblerful of water every morning on rising. The nitrate tends to dilate the vessels like the nitrites, but it acts more slowly and more persistently.

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Angioma. *See* TUMOURS, CAPILLARIES, ELECTROLYSIS.

Angio-neurotic Œdema. *See* URTICARIA.

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I. INJURIES

SPRAINS.—Owing to its position the ankle is a frequent seat of sprains, which are as a rule produced by a mis-step or a fall upon the foot. They vary much in severity. Some are so trivial that their effects soon pass away, while in other cases the sprain is associated with a fracture or a dislocation, one or both, or a displacement of a tendon. Included in the class of sprains of the ankle, therefore, are a variety of lesions, the chief among them being the tearing of ligamentous and other structures. The extent to which ligaments are lacerated will depend upon the severity of the twist or wrench. In some cases a few fibres only are torn, in others the greater part of a ligament may be separated from the bone to which it is attached.

At the time of the accident the patient will experience a sharp, sudden, and often severe pain, frequently referred to the outer side of the foot owing to rupture of some fibres of the external lateral ligament. There will be a certain amount of shock, together with a sick faint feeling, and, unless the injury be a slight one, complete inability to use the limb. The initial pain will probably soon subside, but in the course of a few hours the injured parts become swollen and the seat of a tensive pain. Blood is extravasated into the tissues surrounding the joint, and no doubt in many cases into the joint-cavity itself. The injury will be followed by an attack of synovitis of more or less severity. The swelling due to the extravasated blood and effused fluids is most apparent on the lateral aspects of the joint and in front. In some cases it may be very extensive, and occasion such marked deformity as to suggest displacement of the foot.

Injury to a nerve is indicated when there is severe pain present, extending along the course of its distribution.

Complicating a sprain of the ankle there may be a fracture of some portion of the articular surface, an injury which has been described as a "sprain fracture." A piece of bone or of cartilage is chipped off at the time of the accident, or its nutrition has been so disturbed that it subsequently becomes detached by a process of "quiet necrosis" and forms a loose body in the joint. This additional injury is scarcely ever made out at the time of the accident. It probably explains the ankylosis which sometimes follows what is to all appearances a simple sprain.

Another complication occasionally met with, displacement of a tendon, or tendons, will be described later on. It is of importance, as a possible cause of continued lameness after a sprain of the ankle.

The prognosis in a sprain of the ankle will depend upon the severity of the injury, the amount of damage which the ligaments and other structures sustain, and upon whether treatment has been adopted early and is efficient. A severe sprain often eventuates in the formation of adhesions which leave the joint stiff and its movements embarrassed.

Treatment.—When the patient is seen soon after the accident, the ankle should be wrapped in cotton wool, and a flannel or elastic-webbing bandage firmly applied. This will prevent much effusion and moderate the amount of swelling, while at the same time it gives the patient a sense of relief. Unfortunately, the case rarely comes under observation during this early stage; not until the swelling has attained considerable dimensions.

Then the usual treatment is to apply cold, or what is often much better, hot fomentations. Cold can be applied in the form of an evaporating lotion or as an ice bag, while the limb is kept at rest on a pillow or a splint. In the milder cases, three or four days of this treatment will be sufficient; it can then be replaced by a supporting apparatus, and the patient allowed to get up.

When cold increases the pain, the application of heat gives great relief, and should certainly be adopted.

Whether cold or hot applications are made they should be replaced, usually after a few days, by pressure and massage. By these means the absorption of effused fluids is assisted and the restoration of function encouraged and brought about.

When the sprain is severe and there is reason to suspect extensive laceration of ligamentous structures, the best results are obtained by fixing the joint in an immovable apparatus, such as a plaster-of-Paris bandage, for a limited period, usually *three or four weeks*. This treatment will secure absolute rest for the joint, and make it possible for the patient to get about with the aid of a crutch or stick.

The value of *massage* in the treatment of ankle-sprains can hardly be over-estimated. The main question is, when should it be resorted to? Many surgeons advocate the adoption of massage from a very early period; a few days from the time of the accident. An extensive trial of this method enables us to recommend it very highly in the less severe cases. It will yield a satisfactory result in a much shorter time than any other form of treatment. In severe cases massage should be adopted only after the treatment by cold or heat has been carried out for days, and then it must be combined with the use of a supporting bandage. *If the limb has been placed in an immovable apparatus*, care must be taken not to keep it fixed too long, else adhesions will form, and much trouble and annoyance will be caused. There can be but little doubt that in the past fixation of joints after injury has been carried too far, and the resulting stiffness has occasioned much disappointment. Irregular practitioners, called "bone-setters," have gained much notoriety by their treatment of such cases, the breaking down of adhesions having often restored mobility to a joint which had become quite useless. The important point is, therefore, when should a joint be moved which has been the seat of a traumatic synovitis? Much harm attends too prolonged immobility. It will be found that passive motion is certainly indicated when the swelling has subsided, and the temperature of the part, as estimated by the hand or surface thermometer, has fallen to the level of, or, as sometimes happens, is actually below, that of the corresponding uninjured joint. Pain will be felt when passive motion is first adopted, and this has often been the signal for the patient to discontinue the treatment, and some amount of firmness is necessary to insist on its being properly carried out. The result must, however, be carefully watched. In tuberculous subjects, especially children, a sprain of the ankle is not infrequently the forerunner of a chronic disease, leading to thickening of the synovial membrane, and, if neglected, to complete disorganisation of the joint.

The ankle-joint, after a sprain, may remain for a considerable time stiff and swollen, or it is weak and painful. Fixation for too long a period with the formation of adhesions is usually the cause of a stiff ankle, and the conditions can be overcome by the judicious employment of massage with systematic use of a douche alternately of hot and cold water, or, if obtainable, of tepid sea-water. A stimulating embrocation will also be useful.

More or less permanent weakness of the ankle is also a very troublesome consequence, and may lead to a repetition of the sprain. Massage and judicious use of the joint will usually, after the lapse of some weeks or months, bring about a cure. Meanwhile the ankle will require some sort of support—either an anklet, or, what answers better, an elastic webbing bandage applied figure-of-eight fashion around the joint. Cycling may also be advantageous.

For painful areas, which generally correspond with the attachment of ligaments, much relief will follow daily douching of the part and the rubbing in of an ointment composed of equal parts of iodide of potassium ointment and lanolin.

Dislocation or Displacement of the Peroneal Tendons.—This lesion, although not at all common, sometimes complicates a sprain of the ankle. It should be suspected and looked for when the effects of an ordinary sprain are slow in passing away, also when the patient complains of sudden twinges of pain about the outer ankle whilst walking or standing. When once the luxation has taken place, the slightest cause will reproduce it. It occurs five times as often in men as in women. The two theories in regard to its production may be stated as follows. When the foot is in a position of adduction, a violent contraction of the peroneal muscles will rupture the sheath of the tendons and allow them to escape forwards. According to a second theory, the sheath is torn during a violent muscular effort made to rectify the position of the foot twisted inwards during a fall. In the one case the foot would be in the state of abduction, and in the other, of adduction while the displacement is brought about. In the examples on record, both conditions have been observed.

The peroneus longus appears to be the tendon more frequently dislocated. Of seventeen examples, eleven were of the peroneus longus alone; while in six, the two peronei were displaced. The displacement can only take place in one direction, and that is forwards. Generally the tendon or tendons lie on the outer surface of the external malleolus, in some cases reaching as far forwards as its anterior border.

The symptoms attending this accident are, sharp pain behind the outer ankle combined with more or less loss of power in the limb. In some cases a sudden stoppage in walking without very much pain. Generally the pain disappears with the reduction of the tendons, leaving a soreness which often lasts for weeks. In addition to the pain there is a swelling often limited to the track of the tendons, but sometimes occupying also the outer side of the heel. It is made up of extravasated blood and effusion into the torn sheath. It generally begins to subside in a few days, especially if pressure is employed; some hardness may, however, remain for weeks or months. There is, as a rule, no difficulty in recognising the condition. The one conclusive sign is the presence under the skin covering the external malleolus of the displaced tendon or tendons. When the case is seen soon after the accident, the tendons can readily be felt rolling under the fingers. If the swelling has appeared, some amount of pressure will have to be exercised before they can be recognised. The displaced tendons are easily pushed back into their normal position, especially when the ankle-joint is extended. Displacement of the peroneal tendons may complicate a sprain, or it may be the chief lesion. It also sometimes complicates a fracture of the lower end of the fibula. The condition is likely to be mistaken for synovitis of the tendinous sheath, sprain of the ankle-joint, or fracture of the external malleolus. The presence of the tendons in an abnormal situation, with the absence of symptoms indicating the other lesions, will make its recognition easy.

Treatment.—The indications are to reduce the tendon, and to maintain it in position until the torn structures are healed. There is, as a rule, no difficulty in reducing the tendon, which can be done by extending the foot, abducting it, and pushing the tendon back into its proper place. Suitable compresses are then applied, and are kept in position by a bandage firmly applied. Absolute rest in bed, with the foot raised, must be observed. After a fortnight of this treatment a silicate bandage or some similar immovable apparatus is to be applied, which will allow the patient to move about. In a month, if the reduction is maintained, walking may be permitted, but it must be done cautiously and only for short periods at first. The difficulties in maintaining the tendon in position is in some cases very great, if not impossible. It has been proposed to divide the tendon in such cases. In a case of this kind the luxated tendon of the peroneus longus was divided under the cuboid, the torn sheath irritated by the introduction of a tenotome, the tendon replaced and the limb fixed. In three weeks the patient was pronounced cured, and after five years the cure remained perfect. An interesting case of dislocation of the peroneal tendons is published (*British Medical Journal*, 1897, p. 1216), in which a novel procedure was adopted. The sheath of the tendons was exposed and the rent in it stitched. An oblong piece of periosteum, covering the outer surface of the fibula, was then peeled off, left attached to the posterior margin of the bone, and fixed securely to the fascia behind the sheath. In this way a piece of periosteum arched over the peroneal tendons. Three sutures were now put into the sheath, which fastened it to some firm fascia behind the groove. The incision healed, the leg was put up in plaster of Paris for some weeks, afterwards a leather support was worn. The cure appears to have been permanent. This plan of treatment is of great value when the tendons refuse to remain in their proper position and other methods of treatment are of no avail.

The tendons of the tibialis posticus and of the tibialis anticus may also be displaced. In the case of the former, the luxation produced by a fall was diagnosed, reduced, and the foot fixed for three months.

WOUNDS of the ankle are divided into two classes: those that penetrate the joint and those that do not. The latter are of but slight consequence unless attended with suppuration, which may extend to and endanger the articulation. Wounds which communicate with the interior of the joint must always be regarded as serious accidents, and will require careful treatment. Neglect of proper precautions will lead to destruction of the joint, and possibly to a fatal issue.

The majority of penetrating wounds arise from gunshot injuries. Some, however, are produced accidentally by sharp tools, such as chisels, falling on the foot. The symptoms vary; usually there is an escape of synovia, in the form of a glairy, oily fluid, which appears more or less mixed with blood. The quantity may be scarcely appreciable if the aperture through which it escapes is small. It is soon followed by swelling of the parts from hæmorrhage and effusion into the joint. When the parts are not infected, and suitable treatment is provided, the synovitis which comes on will not be severe and will readily yield. After absorption of the effused fluids, the functions of the joint, after a time, are re-established, there being but little impairment of movement. When, however, micro-organisms are introduced with the instrument inflicting the wound, an acute septic arthritis will generally follow (*vide* "Ankle-Joint, Diseases of"). The case will almost at once assume a serious character, and the prognosis, both as regards limb and life, may be extremely grave.

As it may be often very difficult to determine with certainty whether a wound has penetrated the ankle-joint, it is always wise to treat all wounds in this part as if they penetrated the joint, to clean and close the opening, to apply an antiseptic dressing, and to immobilise the part.

WOUNDS—GUNSHOT.—Such accidents, rare in civil practice, are always serious, but in no class of cases does modern surgery find a more important field or hold out brighter prospects of success. There is so little subcutaneous tissue about the ankle that in all gunshot injuries of this region, beyond a mere graze, important structures must inevitably be damaged. Tendons may be divided and their synovial sheaths opened. The nerve and vascular supply to the foot may be cut off. The bones, in all probability, will be comminuted, and not only the ankle-joint itself, but the articulations of the foot may be opened. Should septic or infective organisms gain access to such a wound, and the patient survive the subsequent toxæmia, disablement, more or less prolonged, must follow. The general health will be taxed by prolonged suppuration and hectic fever. Locally the consequent arthritis and necrosis, together with the non-union of divided nerves and tendons, will result in a deformed, ankylosed, and perhaps useless limb. But, judging from the experience gained in recent years, and from the brilliant results following the antiseptic treatment of compound fractures extending into the ankle-joint from other forms of injury, there can be no doubt that, in the future, conservatism will replace amputation, formerly so universally adopted in the treatment of all gunshot injuries of the ankle, whenever the extent of the injury does not otherwise preclude all hope of recovering the functions of the limb.

Treatment.—Every effort must be made to obtain healing without suppuration. Asepsis and immobilisation of the foot at right angles to the leg are the all-essential principles. When a projectile has passed right through the ankle, the wound of exit must be explored, and the degree of fragmentation of the bones determined. The condition of the astragalus especially should be examined, as this bone is apt to be not only comminuted, but actually pulverised, when struck by a bullet at a high rate of velocity. If necessary, the wound of exit must be enlarged to permit of the removal of all loose fragments of bone, and often the whole astragalus had better be sacrificed. The divided ends of tendons and nerves must be, whenever possible, carefully brought together, and re-united by fine silk or chromic gut sutures. The wound is now irrigated with 1 in 30 carbolic lotion, or 1 in 3000 sublimate solution. After provision for drainage has been made, and the part covered with a thick layer of wood-wool, provision for rendering the joint immobile must be arranged, and in such a manner that any change of dressing does not involve disturbance of the parts. Should conservative treatment have been attempted and have failed, secondary excision may be practised in suitable cases, and is attended with considerable success. In the majority of such cases, however, the extent of the local mischief, or the patient's general condition will call for secondary amputation, and fortunately the mortality of this operation is small.

Primary amputation is demanded when the soft parts are extensively torn away, when there is extensive comminution or fissuring of the bone, and especially when the fissuring of the tibia and fibula extends beyond the malleoli up the shafts of these bones, and when at the same time the posterior tibial vessels or nerve are destroyed. No more of the limb than is necessary should be removed, and Syme's amputation or disarticulation of the foot at the ankle is to be preferred whenever possible.

Ankle-Joint, Fractures in the Vicinity of the.—The most important as well as the most common of these fractures is the one with which the name of *Pott* is associated. It is produced by forcible eversion and abduction of the foot, often the result of a slip off the kerbstone. Occasionally it takes place by forcible inversion and adduction. Usually the astragalus is forced against the inner surface of the external malleolus. In consequence of this impact the fibula first bends and then breaks at its weakest part, which is about three inches above the tip of the malleolus. Besides the fracture of the fibula, the internal lateral ligament is ruptured or the internal malleolus itself is torn off. In most of these cases a very characteristic deformity is met with. The foot is displaced outwards, its outer border is raised, while the sole of the foot looks downwards and outwards. As the upper end of the lower fragment of the fibula is forced towards the tibia there is a marked depression in the situation of the fracture.

In *Dupuytren's fracture* the interosseous tibio-fibular ligament is ruptured or the tibial margin to which it is attached is torn off. The foot, taking with it the lower end of the fibula and the lower outer edge of tibia, which has been separated, is displaced upwards and backwards, while on the inner side, either the internal malleolus or the ligament will give way. The foot is also displaced outwards to a considerable extent, and only to a slight degree everted. There is very marked prominence of the inner ankle which is nearer the sole than normal. There is a very appreciable increase in the distance between the two malleoli. The length of the leg, measured to the tip of the outer malleolus, is diminished.

In these two fractures, besides being displaced outwards, the foot has a decided tendency to slip backwards, occasionally to such a degree that the body of the astragalus lies entirely behind the tibia. When a Pott's fracture is left untreated, and union takes place with the characteristic deformity unreduced, the backward displacement of the foot is very noticeable. This can be readily shown by mapping out the outline of the lower part of the fibula, when it will be found that the portion of bone below the fracture forms an angle with the shaft. Sometimes the ankle-joint is laid open, the fractured internal malleolus coming through the skin, or an unreduced displacement may produce sloughing of the skin covering the lower end of the tibia, in consequence of the pressure exercised upon it.

Fracture of the External Malleolus alone is produced by an inward twist of the foot. The fibula is broken at a lower point than in a Pott's fracture, from one inch to an inch and a half from the tip of the external malleolus. Occasionally, instead of a fracture of the lower end of the fibula, separation of this bone from the tibia is brought about. This accident should be suspected when pain is produced on pressure over the tibio-fibular joint. The tip of the internal malleolus is also in rare instances broken off by the pressure of the astragalus against it, in a twist of the foot. No special treatment is required for these minor and comparatively unimportant fractures; the foot is to be kept at rest for three or four weeks, and when the patient begins to move about, some means must be taken to prevent another twist of the joint. The sprain of the ankle which accompanies the fractures should be dealt with in the manner already described.

Treatment of Pott's fracture.—The indications are to reduce the deformity completely, which can generally be accomplished by extending and adducting the foot while the leg is firmly held, and to adopt suitable means for keeping the fragments in position until union is effected. Various splints are employed. A pair of Cline's, with or without a back splint; a Dupuytren's or internal lateral splint, the notched lower end projecting beyond the foot. Between the splint and the leg a wedge-shaped pad is placed, the base of it extending down almost as far as the internal malleolus. The foot is well adducted and secured to the splint by means of a bandage which is carried up the leg as far as the knee. Some surgeons employ Syme's anterior horse-shoe splint, which may be used in conjunction with a Dupuytren. Others obtain good results with a Roughton's outside splint. Good results have also been obtained by encasing the limb in splints *at once*, and allowing the patient to move about from the beginning.

The important thing in treating a Pott's fracture is to *be quite sure the displacements are properly reduced before the limb is fixed.*

It is a good plan not to keep the splints applied too long—no longer than ten days—before they are taken off, and the ankle-joint gently moved, while the lower part of the leg is firmly held. The splints are reapplied, or the limb encased in a Croft's plaster-of-Paris bandage. Passive movements must be carried out twice a week, and at the end of six weeks the patient will be able to put some weight on the limb. A certain amount of swelling and stiffness will remain for some time; these are treated by the use of the douche and massage.

When in a Pott's fracture the deformity is left uncorrected and union occurs, the utility of the limb will be seriously interfered with. Directly the patient attempts to walk, the eversion of the foot becomes more pronounced and much lameness follows. Moreover, the prominent lower end of the upper fragment of the tibia presses unduly on the skin covering it, and may induce ulceration, and the bone in rare instances projects through the skin. This condition can be remedied by operation. The fibula is divided at the seat of original fracture, and if this is found to be insufficient, the internal malleolus is divided, or the lower end of the tibia is removed, and, if necessary, with it a portion of the displaced (luxated) astragalus. The position of the foot can now be rectified. When the parts have healed, material improvement in progression is observed, a constant source of trouble and pain in the thick projecting osseous mass on the inner aspect of the ankle having been removed.

Dislocations at the Ankle-Joint (Tibio-Tarsal Dislocations).—The entire foot may be displaced in four principal directions, backward, forward, outward, and inward. To these a fifth is added, in which the foot is forced upwards between the tibia and fibula. These dislocations are seldom complete, and very rarely occur without fracture of the tibia, or fibula, or of both bones. They are therefore more appropriately described as fracture-dislocations.

(*a*) *Antero-posterior dislocations.*—1. Backward. 2. Forward.

1. Backward luxation, generally produced by extreme plantar flexion which tears the anterior and lateral ligaments, and the foot is displaced backward. The astragalus is fixed behind the tibia and the articular surface of the latter bone rests upon the scaphoid. The fibula is broken some two or three inches above the tip of the external malleolus. The internal malleolus may also be fractured. Incomplete displacement of the foot backward is frequently found in Pott's fracture.

In the backward dislocation the heel is lengthened and the foot shortened as measured from the malleoli to fixed points in front. The malleoli are nearer the sole than normal, and motion at the ankle-joint is abolished.

The astragalus, if it can be made out, which is difficult when there is much swelling, will be felt between the tibia and the tendo Achillis.

Reduction—flex the leg; heel to be drawn forward and the tibia pushed back. Division of the tendo Achillis may facilitate reduction.

(*b*) *Forward dislocation*, rare. It is usually partial, and one or both malleoli are broken at the same time. The accident is produced by exaggerated dorsal flexion or by direct pressure forward upon the heel. The space in front of the tendo Achillis is filled by the lower end of the tibia, and in front of this bone the trochlear articular surface of the astragalus can be detected. The clinical signs of this injury are—diminished prominence of the heel, with lengthening of the foot. The malleoli will be found nearer the ground and the normal movements of the ankle-joint are abolished.

Much difficulty has been experienced in the reduction of this luxation. Some assistance may be derived by division of the tendo Achillis.

(*c*) *Lateral dislocations.*—3. Outward. 4. Inward.

3. *Outward*, produced by forcible eversion of the foot. It is generally partial and combined with fracture through the lower third of the fibula

and other lesions when it constitutes a Pott's fracture. Most of the examples, therefore, of displacement outward of the foot are to be regarded as instances of fracture-dislocation. It is just possible to meet with a simple dislocation outward of the foot unaccompanied by fracture of the fibula.

Reduction generally easy. When the knee is flexed, adduct the foot and press the tibia outwards.

4. *Inward luxation* rare, produced by violent inversion of the foot, or by direct violence, such, for example, as the passage of a cart-wheel over the ankle. The internal malleolus is commonly broken by the astragalus being forced against it, the line of fracture extending in an upward and inward direction. The external lateral ligament is torn, or the tip of the outer malleolus is fractured and dragged inwards. The deltoid and inferior tibio-fibular ligaments remain intact. The astragalus, which may or may not be broken, is rotated in such a manner that its upper articular surface lies below and in front of the external malleolus. The foot is inverted and its inner border considerably raised.

Reduction is effected by flexing the knee, extending the foot and pressing it outwards. The parts are kept in position by means of a back and two lateral splints. Gentle passive motion at the end of a month.

5. *Dislocation upwards*, Nelaton's dislocation, rare; generally produced by a fall from a height on the sole of the foot. The tibio-fibular ligaments are completely ruptured, and the astragalus is forced up between the two bones which are widely separated. The anterior and posterior ligaments of the ankle are also ruptured, while the lateral ligaments suffer only to the extent of rupture of some of their fibres.

The clinical signs are eversion of the foot, shortening of the leg from the malleoli being nearer the ground. The inner malleolus forms a very distinct prominence on the inner side, with the skin tightly stretched over it. The distance between the malleoli is increased.

In some cases considerable difficulty has been encountered in bringing the foot into its proper position. Generally, however, it can be done by extension combined with, if necessary, a to-and-fro movement to disengage the astragalus which is jammed in between the leg bones. The after-treatment is the same as for the other dislocations.

COMPOUND DISLOCATION OF THE ANKLE-JOINT is always a serious injury, and may necessitate primary amputation of the leg. Under improved wound treatment, the need for such an extreme measure does not now so frequently arise. In deciding whether amputation is required or not, attention must be paid to the nature of the wound, the condition of the soft parts, the age and constitution of the patient.

Amputation will be necessary, if in addition to the dislocation there is much laceration of the soft tissues with splintering of the bone. The accident most likely to produce this state of matters is a heavy weight passing over the ankle.

When the wound is small, by the application of an antiseptic dressing, after the parts have been subjected to careful cleansing, it is possible to obtain rapid healing, the dislocation being thus converted into a simple one. Absolute rest must be enjoined by fixing the limb on a back splint, and later in plaster of Paris. Should suppuration take place, means must be adopted for providing free exit for the pus, and for draining the joint. It has been suggested that, as the malleoli interfere with efficient drainage, they should be removed.

The general plan of treatment in a case of compound dislocation of the ankle when amputation is not considered advisable, is to administer an anæsthetic, to reduce the dislocation, to clean the parts thoroughly with an antiseptic lotion, carbolic acid 1 in 20 or sublimate 1 in 1000, to provide proper means of escape for the effused fluids, to envelop the joint in a wood-wool dressing over sublimate gauze, and to fix the limb securely in suitable splints—generally a wooden back splint, with two interrupted side splints, or to use Paul's splinting with plaster of Paris, the lateral aspects of the ankle being left exposed.

Fractures of the Astragalus are generally produced by falls upon the feet; the line of fracture extending through the neck or the body of the bone. Displacement of one of the fragments frequently takes place. It is often very difficult to make out the precise nature of the injury; in these cases the X rays will afford valuable assistance, by showing us the direction of the fracture, and the character and extent of the associated displacement.

When the fracture is simple with little displacement, the limb should be placed on a back splint, and later encased in plaster of Paris.

Passive movements to be carried out after a month of the treatment by rest.

Should the fracture be compound or the displacement of the fragments be such as to threaten the vitality of the overlying skin, then removal of the bone would be necessary, and the functional results after this operation are very good.

Dislocations of the Astragalus.—In these the astragalus is separated, either partially or wholly, from all its normal connections. It therefore loses its natural relations with the bones of the leg, and with those of the foot, and is carried forward or backward with or without some lateral deviation.

The cause is generally a fall from a height or a sudden twist of the foot.

Forward and outward is the most common variety of luxation. It may be partial or complete. When complete the astragalus is quite separated and carried forward, its head resting on the outer cuneiform and cuboid bones, where it can be seen and felt. The limb is shortened, the foot is adducted and inverted, and the external malleolus prominent. The skin is tightly stretched over the displaced bone and may give way, the head of the astragalus then appearing in the wound.

In the *forward and inward* variety, the astragalus will be found in front of or below the inner malleolus.

Backward.—The astragalus is carried directly backward, or backward with deviation to either side. In some cases the astragalus is fractured, the posterior fragment only being displaced backward.

The bone can be felt projecting between the tendo Achillis and the tibia, while its absence from its normal position can also be ascertained.

Treatment.—Reduction often difficult in dislocations of the astragalus and hardly possible except in the incomplete varieties. The patient is anæsthetised, the knee flexed, and traction made upon the foot, while suitable pressure is exercised on the displaced bone.

When reduction is found to be impracticable, removal of the astragalus is the best treatment. This is also the proper course to pursue when the dislocation is compound.

Sub-astragaloid Dislocations of the Foot.—In these dislocations, the astragalus remains in its normal position, while the scaphoid and os calcis, taking with them the rest of the foot, are displaced from it. The displacement may take place in several directions, *backward, forward, outward* or *inward* and *backward.*

Fracture of the astragalus or of the external or internal malleolus sometimes met with as a complication. The interosseous ligament between the astragalus and os calcis will be ruptured. There is also more or less laceration of the astragalo-scaphoid ligament and the lateral ligament of the ankle-joint. Nearly half of these injuries are compound, and most of them are incomplete so far as the calcaneo-astragaloid joint is concerned.

The most common variety is that in which the foot is displaced laterally, either inwards or outwards, and also carried backwards.

In the backward displacement the heel is lengthened and the front of the foot shortened. An important point of distinction between this accident and dislocation backward of the ankle is to be found in the relation of the tibia and fibula to the scaphoid and cuboid bones. In the sub-astragaloid dislocation of the foot, the rounded and prominent head of the astragalus will be found lying between them. A certain amount of passive flexion and extension of the ankle-joint may still be possible.

Sub-astragaloid dislocation of the foot forwards is a very rare accident. In it the projection of the heel is lost, while the length of the dorsum of the foot is increased. The rounded head of the astragalus can be made out, and is in its normal position, and in front of it there is a depression. Some amount of passive movement possible is in the ankle-joint.

Sub-astragaloid luxation of the foot inward is generally associated with some amount of backward displacement. The distance between the heel and the malleoli is increased, and that between the inner malleolus and the scaphoid is diminished. The outer ankle is unduly prominent, the inner lost in a deep depression, the foot is twisted and presents the appearance of talipes varus.

When the foot is luxated backwards and outwards, the foot is everted, the inner ankle is prominent, and the outer buried in a depression. The foot assumes the position of talipes equino-valgus.

Reduction by traction and manipulation, the patient being anæsthetised and the knee flexed. In a case recently under observation of backward and inward sub-astragaloid luxation of the foot, reduction was easily accomplished by drawing the heel forward and abducting the foot.

When the dislocation is compound, the condition of the soft parts may render amputation necessary or possibly removal of the astragalus alone may be sufficient.

FRACTURES OF THE OS CALCIS, may result from a fall upon the feet or from inordinate contraction of the muscles acting through the tendo Achillis. When due to muscular action the posterior part of the bone is separated and displaced upwards. Means must be taken to retain the fragments in apposition, which can usually be done by flexing the knee, extending the foot, and immobilising the parts. The limb should not be used for a month, and then only with great care.

Sometimes the bone is broken into many fragments, the fracture also implicating neighbouring bones. When it is simple a good result is generally obtained. If there is a wound and it is allowed to get septic, more or less necrosis is almost certain to follow. The treatment must be conducted on ordinary lines.

RUPTURE OF THE TENDO ACHILLIS.—This is produced by a sudden violent muscular contraction, such as occurs when a person makes a supreme effort to prevent himself from falling. The accident is attended by a sharp pain, which gives the patient an idea that he has been struck on the part. A distinct gap will be found at the point of rupture, which is usually about an inch and a half above the insertion of the tendon into the os calcis.

Means must be taken to keep the torn ends of the tendon as near together as possible. This can be done by flexing the knee and extending the foot, this position of the limb being maintained by passing a cord through the heel of a slipper and securing it to a leather collar placed round the thigh.

Provided absolute asepsis can be secured the ends of the tendon should be sutured with chromised catgut. A more satisfactory result may be obtained in a shorter time by following this treatment.

II. DISEASES

N.B.—*The surgical pathology of joint affections will be found in article "Joints."*

Simple Synovitis.—Acute synovitis is of frequent occurrence in the ankle-joint. It may be due to sprains or wrenches of the joint, to excessive use of the limb, or possibly to external cold. For symptoms and treatment see "Sprains of the Ankle."

Septic Synovitis.—Serous effusion into the ankle-joint is occasionally met with in the course of gonorrhœa, syphilis (secondary stage), acute specific fevers (especially scarlatina and enteric fever), and pyæmia. Such cases are sometimes classed together under the term *Septic Inflammations of the Synovial membrane.* They appear to be due to toxines produced by bacterial action, which circulate in the blood and impair the nutrition of various parts of the body. Some of these cases will be mentioned later when speaking of the joint-complications of the diseases in which they occur.

It is necessary to mention that occasionally gonorrhœa, the acute fevers, and especially pyæmia, give rise to suppurative arthritis, an affection which is quite distinct from septic synovitis.

Acute Suppurative Arthritis.—Acute suppurative arthritis of the ankle may arise from one of the following causes:—(1) Wounds of the joint, which give access to pyogenic organisms. (2) Pyæmia. (3) During the course of scarlatina, enteric fever, small-pox, gonorrhœa, and puerperal fever, in all of which it is probably of pyæmic origin. (4) By extension of acute suppurative inflammation—infective periostitis or osteomyelitis—from the bones of the leg. The acute arthritis of infants, which sometimes involves the ankle-joint, commences as an acute inflammation of the growing tissue of the diaphysis immediately beneath the epiphysial cartilage; pus forms rapidly and may extend to the joint. (5) By extension of suppuration from the surrounding soft parts.

In the earlier stages of the disease the synovial membrane is inflamed, but later it takes on the characters of granulation-tissue. The cartilages become softened, eroded, and more or less destroyed. The ends of the bones, when exposed by destruction of the cartilage, become the seat of rarefactive osteitis. The ligaments are infiltrated and softened, and the surrounding parts are likewise involved in the inflammatory process.

The onset of this affection may be marked by a rigor. The local signs are rapidly increasing swelling, heat, redness, and severe pain. Any movement gives rise to intense suffering. The constitutional symptoms are those of very severe fever. After a time the presence of pus may be recognised by œdema of the skin and fluctuation. Unless effective treatment be adopted, the patient will probably sink rapidly, and die from exhaustion and general blood-poisoning.

Treatment in these cases must be carried out promptly. The limb should be kept absolutely at rest. As soon as fluid is detected, it should be removed by an aspirator, and, if found to be purulent, free incisions should be made, the joint-cavity well washed out, and a drainage tube inserted. Subsequently boracic fomentations should be applied and irrigation with weak antiseptic lotion should be practised at intervals of not more than four hours. This treatment will sometimes prove effective in arresting the disease, but if, in spite of it, the local and constitutional symptoms increase in severity, no time should be lost in resorting to amputation. In most cases it will be sufficient to remove the foot by Syme's method, but occasionally amputation through the lower part of the leg will be advisable.

AFFECTIONS IN THE COURSE OF THE ACUTE-SPECIFIC FEVERS.—During the course of *Scarlet Fever* the following joint-affections may arise: (1) Scarlatinal Synovitis. This occurs most often at the end of the first, or the beginning of the second week of the fever. Most frequently the wrists are the seat of the trouble, but the ankles are occasionally involved. Complete recovery usually takes place. The affection has been supposed to be of the nature of acute rheumatism, but more probably it is due directly to the scarlatinal poison, or to the action of septic material absorbed from the throat. (2) Acute Suppurative Arthritis, of pyæmic origin, may also occur. It is rarer than the scarlatinal synovitis, and the ankle-joint is only occasionally involved. (3) True rheumatic arthritis is a still rarer complication.

Somewhat similar affections may occur in *Enteric Fever*, and the other acute fevers.

TUBERCULOUS ARTHRITIS.—Tuberculous disease is of frequent occurrence in the ankle-joint, and is a grave affection because of its liability to extend to the surrounding structures. Most frequently the synovial membrane is the seat of origin of the disease, but in some cases the joint-affection is secondary to tuberculous disease of one of the malleoli, of the astragalus, or even of other bones of the tarsus. The astragalus is the bone which is probably most often attacked primarily. Occasionally the primary deposit of tubercle takes place in the neighbouring tendon-sheaths, and the joint is invaded by extension.

Whatever may be its seat of origin, the tuberculous process, when once the joint is involved, is very apt to extend rapidly to all the structures of the articulation. The synovial membrane becomes greatly thickened, the cartilages are gradually destroyed, and the ends of the bones, which are thus exposed, fall into a state of caries. Unless the disease be arrested by proper treatment, suppuration will probably occur, and the case will progress from bad to worse.

Symptoms.—The most important symptom undoubtedly is swelling, which as a rule first appears in front of the malleoli, at either side of the extensor tendons. Fulness also appears at the back of the joint, thus obliterating more or less the hollows on either side of the tendo Achillis. Later, the swelling becomes general, extending all round the malleoli and masking the extensor tendons. Wasting of the muscles of the calf occurs at an early stage and is often very marked. We are of opinion that muscular wasting takes place more rapidly in tuberculous disease than in most other joint-affections, and that it is a point of diagnostic importance in doubtful cases. The movements of the ankle are more or less impaired. This, however, may only be noticeable when the joint is fully flexed or fully extended, the middle ranges of movement being almost perfect, that is, if the articular surfaces are not involved. Walking is interfered with to a greater or less degree. The amount of pain varies very much, and gives little help in arriving at a diagnosis. The last remark also applies to the presence or absence of local heat and tenderness. When the joint-lesion is secondary to tuberculous disease of the bones, the latter are usually greatly enlarged. This is especially marked in the case of the malleoli, but is less easily detected in the case of the astragalus. Howard Marsh places the symptoms of tuberculous disease of the ankle-joint in the following order of importance: (1) Swelling; (2) Limping; (3) Wasting of the calf-muscles; (4) Stiffness of the joint; (5) Heat of the surface; (6) Pain; and (7) Tenderness. When suppuration occurs the pus gradually makes its way to the surface, and points either in front of, or less frequently behind, one of the malleoli. Occasionally it bursts into the tendon-sheaths, and tracks widely along them.

Diagnosis.—The diagnosis of these cases is sometimes very difficult. They are liable to be confounded with tuberculous disease of the lower ends of the tibia and fibula, of the tarsus, or of the neighbouring tendon-sheaths. Fortunately in the earlier stages the treatment is the same for all these affections. When sinuses are present, the direction which is taken by a probe gives valuable and reliable assistance. In all cases, however, when an operation is necessary, the incisions should be made very carefully, so as to avoid opening joints previously uninfected.

Treatment.—When the disease is seen in the early stage and in the case of children, it can usually be arrested by a proper course of treatment extending over from six to twelve months. Recovery may then take place with little or no impairment of the part. After the age of puberty the chances of success without resorting to operative measures are not so great. *It is absolutely essential that the joint be kept at perfect rest*, and this may be effected by splints of leather, poroplastic felt, or gutta-percha, or by strapping the ankle with mercurial ointment. An excellent method is the use of Paul's splinting with plaster-of-Paris bandages. Care must be taken to keep the foot at a right angle with the leg. Appropriate constitutional treatment should also be adopted. Later, when the disease is quiescent, plaster or silicate bandages may be applied, and the patient allowed to move about on a Thomas's knee-splint, or with the knee flexed on a bucket-and-pin leg.

Other methods of treatment which have been recommended are the injection of *iodoform emulsion* and the production of *hyperæmic engorgement* of the joint by means of bandages. These methods are still *sub judice*.

If, in spite of treatment, the disease progresses, or if suppuration have occurred, the operation of *arthrectomy* should not be delayed. During recent years, when great care has been taken to remove the whole of the infected tissue, very good results have been obtained, but if any tuberculous bone or synovial membrane be left behind, a recrudescence of the disease is to be expected. The details of the operation will be considered later, but it may be stated here that the exact method to be adopted must vary with the extent of the tuberculous process. If the astragalus be much diseased, or if the joint-affection be at all extensive, excision of this bone is indicated, with subsequent removal of the whole of the diseased synovial membrane, and of affected portions of the articular surfaces of the tibia and fibla. The results of this operation, as regards the usefulness of the limb, have been very satisfactory. When it is deemed inadvisable to remove the astragalus, access to the joint may be gained by division of the malleoli at their bases.

In cases of extensive disease, where we cannot hope to get a good result by arthrectomy, it is necessary to resort to *amputation*. Syme's amputation is sufficient in most cases and leaves an excellent stump, but it is open to the objections that the line of section is very near the seat of disease, and that portions of tuberculous material may be left behind. If the tibia and fibula be much diseased, if the suppuration have extended upwards along the tendon-sheaths, or if the heel be riddled with sinuses, it is better to amputate through the lower part of the leg.

SYPHILITIC AFFECTIONS.—The ankle would appear to be the third in the order of frequency with which various joints are affected by syphilis. The knee claims a very large proportion of syphilitic joint-affections, and the elbow a moderate number, whilst the ankle is only occasionally involved. In the secondary stage, *synovitis with effusion* may occur about the same time as the skin eruptions and throat affection. In the tertiary stage the ankle may be the seat of *diffuse gummatous infiltration of the joint capsule*, accompanied by thickening of the synovial membrane and a moderate effusion of synovia. More rarely the joint is affected secondarily to a gummatous osteitis of the neighbouring bones. The importance of recognising these cases from tuberculous lesions is evident. Affections of this joint have also been met with in congenital syphilis.

Treatment by rest and by the internal administration of mercury and potassium iodide will speedily cure the secondary synovitis. In cases of tertiary affections, however, the treatment will be more tedious, and will necessitate a prolonged course of iodides, with appropriate local treatment.

GONORRHŒAL AFFECTIONS.—With the single exception of the knee, no joint is, in our experience, more liable to be affected by gonorrhœa than the ankle. This complication most often occurs during the third or fourth week of the disease. It is usually accompanied by considerable effusion, and in acute cases by severe pain. In most cases it takes the form of a more or less *acute synovitis* with serous effusion, but in a few rare cases there is a *suppurative arthritis*. Serous effusion is probably caused by toxines of bacterial origin, which have been absorbed into the blood from the urethra. Suppurative cases are accompanied by the presence of bacteria themselves in the joint. The gonococcus has been found in some cases. So frequent are gonorrhœal joint affections, that it is always well to consider the possibility of this cause in cases of effusion of doubtful origin. Unless the cause be treated, the articular trouble will probably undergo little improvement. In most cases complete recovery takes place, but occasionally the effusion is of a very plastic character, and gradually leads to ankylosis. In women it is stated that a similar affection may be due to non-specific leucorrhœa, and in children undoubted cases have occurred in connection with gonorrhœal ophthalmia.

A very intractable form of this disease sometimes affects the ankle and tarsal joints simultaneously, and may be accompanied by inflammation of the plantar fascia and other fibrous structures of the sole, thus leading to very troublesome flat-foot. *Plantar neuralgia* is frequently due to gonorrhœal inflammation of the fibrous structures of the sole.

Treatment, in the first place, must be directed to removing the cause. The joint should be kept at rest in the earlier stages, and an attempt made to get rid of the effusion by blistering and a moderate degree of pressure. The internal administration of salol is also very effective in promoting absorption. The joint should not be kept at rest too long, because of the risk of ankylosis, and hence massage and passive movement should be commenced as soon as the greater part of the effusion has been absorbed. It must be remembered, moreover, that relapse is very liable to take place, and hence the patient should continue under treatment until the gonorrhœal discharge has quite subsided.

Osteo-Arthritis.—The ankle-joint is comparatively rarely affected by osteo-arthritis. When the disease does occur, both ankles are usually involved, and generally other joints at the same time. The symptoms are similar to those of osteo-arthritis in other situations—stiffness, pain, gradual impairment of movement, progressive weakness, grating, and the presence of osteophytes. The affection is apt to be accompanied by an intractable form of flat-foot, partly due to muscular weakness, but largely to involvement of the ligamentous structures of the sole.

Treatment may be chiefly conducted as for osteo-arthritis in other situations. Pain will be relieved by rest, elevation of the limb, and hot fomentations. The joint should be kept warm by flannel, and in most cases the substitution of a boot of soft leather or cloth for the ordinary boot will be necessary. Daily massage and friction with liniments will give considerable relief in most cases. Occasionally the use of a light steel support is advantageous.

Gouty Arthritis.—After the great toe the ankle is one of the most favourite sites of gouty deposits. This is probably due to the sluggish nature of the circulation in the lower part of the legs of many of those who are subject to gout. The disease is accompanied by a local deposit of urate of soda, and from time to time the patient experiences explosive attacks of inflammation of the peri-articular structures, although the attacks usually follow a less typical course than in the case of the great toe. The treatment of these cases must be as for gout in other situations.

Tabetic Arthropathy (*Charcot's Joint Disease*).—In some cases of locomotor ataxia a peculiar joint affection occurs, especially in the knees, hips, ankles, and elbows. The ankle is affected in about four per cent of cases which present this complication. It may occur either in the pre-ataxic or the ataxic stage. The onset is usually sudden, and is accompanied by abundant effusion into the joint. As a rule there is but little pain. In favourable cases the effusion begins to be absorbed in the course of a few days, and the joint may return almost, if not quite, to its normal condition. More frequently, however, the effusion is more persistent, and rapid degenerative changes take place, leading to excessive mobility of the joint. Occasionally a considerable amount of new bone is thrown out, and the movements are restricted.

Similar changes are occasionally met with in the foot, owing to involvement of the tarsal bones and joints. The bones, which are frequently enlarged, tend to fall downwards and inwards, giving rise to a condition of flat-foot, the so-called *tabetic club-foot.*

Diagnosis in these cases requires care, because at the time of occurrence of the complication the signs of locomotor ataxia may not be evident to casual inspection. The disease is most liable to be mistaken for rheumatoid arthritis, but, independently of the other signs of ataxia, it can usually be distinguished by the abundance of the effusion in the early stages, by the rapid progress of the disease, and by the freedom of movement in most cases.

Treatment.—In the earlier stages rest upon a splint may do something to check the disease, and it is possible that the removal of the fluid by means of an aspirator may act beneficially. In the later stages the rapid progress may be somewhat stayed by the use of splints. Excision of the joint is to be avoided, and the same remark is usually applied to amputation. Southam, however, has recently recorded four cases of Charcot's disease of the ankle and tarsus in which he has removed the limb with excellent results.

Loose Bodies.—These are very uncommon in the ankle-joint. When they do occur, they may give rise to symptoms similar to those caused by loose bodies in other joints (*vide* "Knee-joint"). Under such circumstances they should be removed through an incision with antiseptic precautions. Some years ago a case came under our notice, in which, as a result of an injury, a portion of the articular cartilage of the astragalus became detached, and formed a loose body in the joint. This was removed, and the patient made a complete recovery.

Ankylosis.—Fibrous ankylosis may occur in the ankle-joint as a result of injury, as a result of one of the forms of septic synovitis, especially the gonorrhœal variety, or from tuberculous arthritis. Bony ankylosis may occur in cases where the cartilages are destroyed by suppurative or tuberculous arthritis.

Treatment.—In cases of fibrous ankylosis some benefit may result from forcible wrenching under an anæsthetic, followed by daily massage and friction with liniments. In severe cases, and in cases of bony ankylosis, no operation, with the object of restoring movement, is likely to be of service, but, if the foot be in bad position, an attempt may be made to rectify the latter.

Hysterical Disease.—The ankle is not infrequently the seat of hysterical joint-disease. In most cases it is said to have arisen in a sprain or other injury, more or less trivial. The diagnosis from organic disease of the joint must rest upon the absence of the objective signs of disease, and, if necessary, upon an examination under an anæsthetic.

Treatment must be directed to improvement of the general health, and to encouraging the patient to use the joint. Blistering, passive movements, massage, and the faradic current may be of service.

Diseases of the Tarsal Bones and Joints

The tarsal-joints may be the seat of simple inflammatory, pyogenic, tuberculous, syphilitic, rheumatoid, or gouty disease; they are also subject to tabetic arthropathy. The tarsal bones are not infrequently the seat of tuberculous deposits, and occasionally of sarcomatous growths. With regard to these affections, except tuberculous disease and new growths, it is unnecessary to add anything to the remarks which have already been made in connection with the ankle-joint.

Tuberculous Disease.—In the great majority of cases tuberculous disease of the tarsus commences in the bones as a tuberculous osteomyelitis, and involves the joints secondarily. The joints are first affected in a few cases. Of the bones the calcaneum is the one which is most often involved, the disease usually starting in front of the posterior epiphysial line, and in many cases remaining for a long time strictly limited to this situation. After a time, however, the astragalo-calcaneal or the calcaneo-cuboid joints may be invaded. With the exception of the calcaneum, the cuboid and the astragalus are the most subject to disease. When commencing in the astragalus, tuberculous disease is most important, as it is very apt to extend rapidly to the neighbouring joints. The scaphoid and the cuneiform bones may also be the primary seat of trouble, and in such cases the disease is apt to spread widely, because these bones enter into the formation of the complicated medio-tarsal joint. The anterior and posterior tibial tendon-sheaths are sometimes invaded. It would appear that phthisis is especially apt to supervene in cases of tuberculous disease of the tarsus.

The *symptoms* presented by these cases are swelling, pain, tenderness, and more or less difficulty in walking. Local heat and redness of the skin are inconstant. The seat of the swelling varies considerably with the exact origin of the disease, but as a rule it is most evident on the dorsum and at the sides of the foot, and is only slightly recognisable on the sole. When suppuration occurs the pus usually points in the same situations.

Treatment.—In the early stage the treatment is practically the same as for tuberculous disease of the ankle-joint. Operative measures, however, must not be long deferred, for, owing to the complicated structure of the tarsal joints, the disease is apt to spread widely, and, moreover, in the early stage the tuberculous process is often limited to one part, and can be cured by a simple operation, whilst in the later stages the whole tarsus is involved, and extensive removal is necessary. Operation should be undertaken at once if there are signs of suppuration, or if the disease spreads in spite of rest and local treatment. It should be a rule to remove too much rather than too little when operating upon these cases. If the whole of the diseased structures be removed good results will follow, but incomplete operations are certain to bring failure. In the case of the os calcis it is often sufficient to gouge out the diseased part, but there should be no hesitation in removing the whole bone if it be extensively diseased. The results of the latter operation have been excellent; in some cases the patients have walked perfectly after the parts have become sound. Disease of the astragalus can seldom be treated satisfactorily by mere scraping, and hence removal of the whole bone is usually necessary. If the disease has spread to the neighbouring joints, the tuberculous synovial membrane must also be taken away. Excision of the cuboid bone has given good results in cases where the tuberculous process has commenced in, and remained limited to, that bone. When the disease has been very extensive, almost the whole of the tarsus has been excised, but usually under such circumstances it is necessary to amputate either by Syme's method or through the lower part of the leg.

Sarcoma.—The tarsus is occasionally the seat of sarcoma, which may be of either the subperiosteal or central variety. In the early stage the diagnosis from tuberculous disease may be very difficult, but the rapid growth will soon enable a definite opinion to be formed. In doubtful cases it may be well to make an exploratory incision. Sarcomata arising in this situation rapidly become generalised, and would appear to possess a higher degree of malignancy than sarcomata arising in the thigh or leg.

The only treatment is amputation at a sufficient distance above the seat of the growth.

Diseases of the Tendon-Sheaths around the Ankle

Tenosynovitis.—The tendon-sheaths in the neighbourhood of the ankle are very liable to inflammation, especially the sheaths of the peronei tendons. *Acute tenosynovitis* frequently arises from excessive use of the foot or from sprains. It usually subsides readily with rest and the application of glycerinum belladonnæ. *Subacute and chronic tenosynovitis* may result from gonorrhœa, rheumatism, or syphilis (second stage). The same treatment, with appropriate constitutional remedies, should be adopted.

Suppurative tenosynovitis is due to the entrance of septic matter into the tendon-sheaths. A free outlet for the pus must be provided, and antiseptic fomentations applied regularly.

TUBERCULOUS TENOSYNOVITIS in this situation usually results in great thickening of the sheaths, with little effusion. The peronei, the extensors of the toes, and the posterior tibial sheaths are the usual situations. The disease may be primary or secondary to tuberculous disease of the ankle-joint or tarsus. In the early stage the treatment must be conducted on the same lines as for ankle-joint disease. Should this fail, as much as possible of the diseased tendon-sheaths should be excised.

DISEASES OF THE BURSÆ.—For the situation and diseases of the bursæ in the region of the ankle-joint, see "Bursæ, Diseases of."

III. OPERATIONS

ERASION OF THE ANKLE-JOINT.—*Arthrectomy.*—The diseased structures are removed by freely opening the joint on its lateral aspects, or by an anterior incision, which severs all the soft structures in front of the articulation. The latter method has the advantage that it brings into view every nook and corner of the joint, and renders removal of diseased tissues much more certain. Its drawback lies in the severance of the tendons, which, however, will unite if they are carefully sutured and sepsis is avoided. After the diseased tissues are removed with knife and scissors, the joint cavity is flushed out with a warm antiseptic lotion, the edges of the incision are brought into apposition without any opening for drainage, and the foot fixed at right angles in plaster-of-Paris. If the astragalus is diseased it must be removed, the results after such a procedure being often very satisfactory.

EXCISION OF THE ANKLE-JOINT.—This is not often performed, and should not be entertained for disease of the ankle unless it is limited to the tibio-tarsal articulation, and only after treatment by rest has been carefully tried. The ankle is also sometimes excised in cases of injury, such as compound dislocation and complicated fracture. The after-treatment is always troublesome and difficult, and the result in many instances unsatisfactory. Access to the joint can best be gained by a free, curved, external incision, extending down between the extensor tendons and the fibula to the outer surface of the astragalus. The periosteum is detached from the fibula and the ligaments separated as they are exposed. Almost an inch of the fibula is now removed, either with a saw or a chisel. The lower end of the tibia is next cleared, and to facilitate this part of the operation an incision is made along the inner surface of the tibia, and in the long axis of the bone the malleolar end of the tibia is removed. Also, as much of the astragalus as is deemed necessary, or frequently the entire bone, is taken away. In the after-treatment care must be taken to fix the foot and leg in a good position. Some form of plaster-of-Paris dressing with Paul's splinting will be found to answer best. The result most to be desired is firm ankylosis.

EXCISION OF THE ASTRAGALUS.—Astragalectomy is sometimes performed for tuberculous diseases; more frequently it is resorted to in cases of injury, such as dislocation, and in inveterate club-foot (Lund's operation). The bone is removed by making a free incision on the antero-external aspect of the ankle-joint, commencing two inches above the ankle at the anterior margin of the fibula, and extending forwards over the outer surface of the astragalus on the outside of the extensor tendons nearly to the prominence of the fifth metatarsal bone. The ankle and mid-tarsal joints are opened, while the body and head of astragalus are freely exposed. The interosseous calcaneo-astragaloid ligament is freely exposed and divided. A smaller incision is now made over the lower end of the tibia, and through it the ligaments which still hold the astragalus in position are divided and the bone seized with a strong forceps and removed through the external incision.

Excision of the Os Calcis may be required in disease of the bone, also occasionally in cases of injury. It can be accomplished by making a longitudinal incision two inches in length, parallel with and along the inner margin of the tendo Achillis, the lower end of the cut being over the prominence of the heel. From this point another incision is carried round the heel and forwards along the outer aspect of the foot to the base of the fifth metatarsal bone. The tendo Achillis is detached, and the joint capsule at the posterior and outer aspect of the os calcis, together with the calcaneo-fibular band of the external lateral ligament, is severed.

The peronei tendons are preserved. The interosseous ligament between the os calcis and astragalus is divided, also the calcaneo-cuboid ligaments, the bone being cleared on its plantar and dorsal aspects.

The tendon of the tibialis posticus is drawn upwards over the sustentaculum tali. The os calcis is seized with a pair of long forceps and forcibly dislodged, and any remaining connections detached, and in doing so the structures on the inner surface of the bone are to be carefully avoided. After the operation the foot is fixed at a right angle with the leg and the heel left exposed.

The results of this operation are most encouraging. Subperiosteal resection, although sometimes practised, is of doubtful utility when the operation is performed in cases of disease.

In children the bone can be removed through the heel incision, the vertical cut being dispensed with altogether.

Arthrodesis, or the artificial production of ankylosis in a flail joint, is sometimes practised on the ankle. It often enables a patient to walk with very little assistance, such as that provided by an artificial muscle, although previous to the operation he was obliged to wear heavy, cumbersome instrumental support. A description of the operation will be found in the article "Deformities."

An account of the amputations at the ankle-joint and neighbourhood will be found under "Operations."

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Ankylosis. *See* Joints.

Ankylostoma. *See* Parasites.

Anorexia and Anorexia Nervosa. *See* Appetite, Hysteria, Neurasthenia.

Anosmia. *See* Smell.

Anthelmintics.—These are remedies which kill or expel worms. The frequency of the occurrence of intestinal parasites, especially in children, and the important nervous and other symptoms often associated with their presence, both in children and adults, are referred to at length elsewhere.

CLASSES.—The remedies used in the treatment of intestinal worms are usually considered in two groups; those which poison the parasites are termed *vermicides*, whether they actually kill them or simply stupefy them, so that they are easily expelled; while those that simply expel them from the bowel (drastic purgatives for the most part) are called *vermifuges*.

ENUMERATION.—The principal *vermicides* are male fern, turpentine, pelletierine (an alkaloid obtained from pomegranate root bark), kousso, kamala, areca, which are all used for the tape-worms; santonine, which is used for round worms and thread worms.

The principal *vermifuges* are cathartic purgatives, such as jalap, which may be given alone or a few hours after a dose of a vermicide.

Enemata of salt and water, turpentine, perchloride of mercury, infusion of quassia, often with the addition of the perchloride of iron, are used to reach the thread worms (ascarides), which flourish in the rectum and colon.

METHODS OF ADMINISTRATION.—Generally speaking, anthelmintics should be given when the alimentary canal is as empty as possible, and should be preceded and followed by a purgative. Thus a patient suffering from tape-worm should take a dose of castor oil in the morning, followed by a day's starvation (a cup of tea or a basin of clear soup may be allowed for breakfast and lunch). In the evening, 30 to 60 minims of extractum felicis liquidum should be administered, preferably in capsules. On the following morning a second dose of castor oil should be taken.

Kousso and kamala are both gastro-intestinal irritants, and should not be administered to pregnant women.

Pelletierine may be used as the tannate, in a dose of 8 grains, followed in two hours by an ounce of castor oil.

For the removal of round worms (lumbrici) santonine is the most efficient agent. For a child of five or six years of age, 2 grains of santonine with 1 grain of calomel may be given every night for two or three nights, to be followed each morning by a purgative dose of castor oil.

The treatment of thread worms (ascarides) may be a troublesome matter if the parasites are numerous. Santonine should be administered internally, as for lumbrici, and in addition, anthelmintic enemata should be made use of. The colon should first be cleansed by an injection of warm water, to which borax or bicarbonate of soda (ʒj. to the pint) has been added. When this has come away, half a pint of bichloride solution (1 in 10,000) should be injected high into the bowel through a catheter, and retained for some time. This may be repeated every second or third night. The folds of the anus should also be kept anointed with mercurial ointment, as this not only diminishes the itching at night, but, by confining the worms to the bowel, lessens the risk of reinfection through the mouth, which is often brought about by the child scratching in the neighbourhood of the anus, and afterwards sucking its fingers. The folds of the genitals should also be anointed, as the scratching not infrequently sets up vulvitis or vaginitis, and in some instances even leads to masturbation.

It is important to bear in mind that cases should not be considered cured until a careful examination of the stools warrants such an opinion.

Anthracosis. *See* LUNGS.

Anthrax. *See* MALIGNANT PUSTULE.

Antimony. *See* TOXICOLOGY.

Antiperiodics.—The medicinal antipyretics, which have received the divisional name of antiperiodics, exhibit a much more specialised action than the other members of the larger group. Their distinguishing feature consists in their power of preventing attacks, or of diminishing their frequency and severity, when given beforehand, and of quelling or mitigating actual paroxysms of certain diseases which are, regularly or irregularly, periodic in character.

Cinchona and its allies form the most prominent members of the group, and owe their antiperiodic properties to the alkaloids quinina, cinchonina, cinchonidina, and others found in them.

Quinina, usually employed in the form of its sulphate, although the chloride is as powerful and much more soluble in water, possesses a most remarkable power over the malarial poison, be it filarial or of a mixed nature; a power far more extensive than that of a mere antipyretic. Preventative either of initial infection or of the actual paroxysms of an attack of malaria, it acts specifically in all malarial cases, but it also is an antipyretic acting as the ordinary members of the class do. In addition to its action upon malarial maladies, it has been found to be very useful in warding off influenzal attacks, given in doses of about five grains each morning whenever signs of the approach of an epidemic of this modern scourge appear, and continued until what time its visitation may terminate. During an actual attack of influenza it does not appear to be of any great service; those who have taken quinina for some time and are attacked by the disease, as a rule suffer less both then and afterwards.

Long after all the actual agents in the causation of malarial symptoms must have been got rid of, and when the chances of fresh infection are impossible, the addition of quinina to other remedies given for many non-malarial ills will aid their action, often to a remarkable extent.

In addition to the cinchona alkaloids, beberina from Bebeeru bark, eucalyptus, and arsenic possess reputed antiperiodic properties.

Uses.—Hardly anything need be added to the foregoing as to the uses of antiperiodics. Quinina and the allied alkaloids act specifically upon the poison of malaria, and also form the best means we have in the remittent fevers of hot countries. Patients suffering from malaria can take with impunity doses of quinina so large as assuredly to cause symptoms of cinchonism in others. This fact is beneficial, as large doses are often required to prove of service. Several of the recent synthetic antipyretics seem to assist quinina in its actions. Salicinum, salicylic acid, and salol serve the same purpose in acute rheumatic attacks as quinina in malaria. Beberina is not nearly so powerful a drug as quinina in malaria. Arsenic occasionally aids the action of other antiperiodics; indeed, it may act successfully when given alone, even after the more special members of the group have failed.

Antipyretics and Antipyretic Measures.

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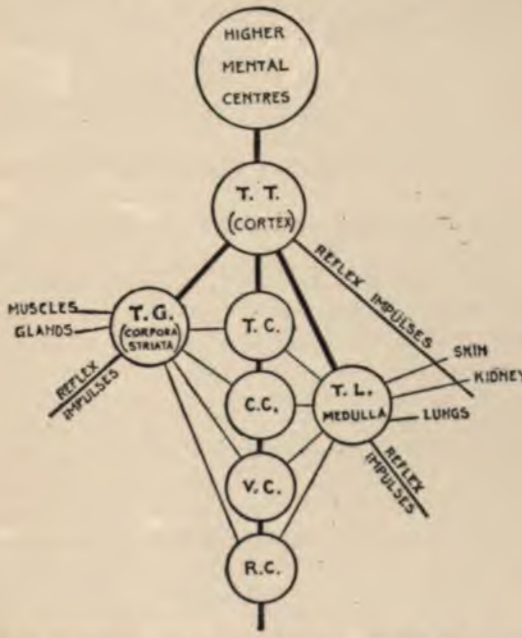
DEFINITION.—(ἀντί, against; πυρέτος, fever)—Drugs or measures which possess the power of lowering increase of body temperature produced by disease. A few drugs can lower the temperature of the body in health, and these only if pushed so far as to cause symptoms of collapse. Measures taken to lower pyrexia are as truly antipyretic as are drugs.

Modes of Action.—Few classes of drugs are used so frequently, and with so small an understanding of their true pharmacological properties, as the class of antipyretics. To properly grasp the important differences which exist between the modes of bringing about diminution of fever by groups of antipyretic drugs, and which are clearly defined one from the other, a word as to the supposed arrangement of the heat-regulating mechanism in the brain will be of service. The temperature of the body is regarded as being under the supervision of a thermotaxic centre, situated in the cerebral cortex, which in turn influences a secondary centre regulating the production of heat, the thermogenetic centre in the corpus striatum, and another similar to it, the thermolytic centre in the medulla, concerned with the amount of heat allowed to pass out of the body. The temperature of the body is regulated by these centres; the production or acquirement of heat being counterbalanced by the loss. The clinical use of the various means of reducing fever is dependent on their action upon one or other of these centres, upon both the thermogenetic and thermolytic central ganglia, or upon their peripheral nervous communications (cf. Schema).

Scheme to illustrate the Central Heat-Regulating Mechanism. Adapted from Hale White.



Centres—T.T., Thermotaxic; T.G., Thermogenetic; T.L., Thermolytic; T.C., Trophic; C.C., Circulatory; V.C., Vaso-motor; R.C., Respiratory.

The muscles both of voluntary and of involuntary movements and the glands are the chief sources of heat-production; the skin, the lungs, and the kidneys the most important agents in heat-dispersal.

The measures adopted by physicians to combat pyrexia may be directed towards a decrease in production, or an increase in loss of heat, or may induce both of these actions. The drugs which increase the loss of heat act chiefly by stimulating the sudorific mechanism; others, as for instance belladonna, while diminishing sweat secretion, by dilating the blood-vessels of the skin increase heat loss by radiation. Cold baths, sponging, and the like, abstract warmth from the body. The thermogenetic centre is influenced directly by a number of remedies, either alone or in conjunction with an accompanying but not so marked action upon the thermolytic centre. The large class of the synthetic products, added to the antipyretic class through modern chemical research, act in this way.

While there are undoubtedly many advantages to be derived from the use of drugs which diminish fever through the medium of the official nervous regulating system, and while the results obtained from them have often been most valuable, they possess certain disadvantages in that, as they act especially upon the nerve-centres for heat regulation, they are apt to influence the normal activity of neighbouring centres.

Synthetic Antipyretics.

Classification of Antipyretic Remedies

A. Decrease Heat Production—
  1. Action on Thermogenetic Centre (Hypothetically).
     Phenazonum, Acetanilidum, and similar drugs. Alcohol (?), Chloral.
  2. On Metabolism principally.
     Quinina, Acidum Salicylicum, Alcohol, essential oils.
  3. Action unknown.
     Cold baths if prolonged.

B. Increase Heat Loss—
  1. Sudorifics, Diuretics, Purgatives, and Alcohol.
  2. Acting on Thermolytic Centre.
     Phenazonum, etc. (slightly), Antimonium.
  3. Physical Methods.
     Cold baths, sponging with water or alcohol, etc. Warm baths. Venesection.
  4. Through the circulation (probably) dilating vessels of skin.
     Antimonium, Chloral, Scilla, Alcohol.
  5. Unknown. Aconitum, Digitalis, Veratrina.

C. Indirect Action—
  1. Removing cause of irritation, physical or mental.
     Purgatives, sedatives, relief of tension, etc.
  2. Specific action on causative poison.
     Quinina, Acidum Salicylicum.

Phenazonum (Antipyrin), Acetanilidum (Antifebrin), and Phenacetinum are the drugs of this series which are most commonly employed. They all depress the action of the heart, phenacetin the least of the three; if they are given in large doses the blood is affected, methæmoglobin being formed and appearing in the urine. Respiration is not altered; the kidneys and skin are mildly stimulated; indeed erythema and urticaria are not uncommon results of their administration. The combination of caffeina with the synthetic antipyretics is often found to be valuable. Caffeina acts chiefly upon the cerebrum, but it also stimulates without slowing the heart, and raises the blood-pressure, but does not interfere with the antipyretic effect of the drug given with it.

The members of the aromatic group of antipyretics probably act upon the heat-producing centre in the corpora striata. They have been said to owe their power to the property of readily forming substitution and addition products. The aromatic compounds resulting from metabolism are increased in the urine after they have been taken, and they may thus help to free the body from such substances. Indeed, neutral sulphates have been recommended to be given as adjuvants with the view of facilitating the excretion of ethereal sulphates. Another point to be remembered with regard to these drugs is the frequent intolerance exhibited by patients to one or other of them. They should never be prescribed save in small doses to any patient whose reaction towards them is not known.

The Application of Cold.—Abstraction of heat from the body by immersion in cold water (in a cold bath the temperature of the water is below 70° Fahr.), by sponging, packing, applying ice, or evaporating lotions, constitutes the most satisfactory method for the reduction of fever through increase of heat-loss. This subject, however, is fully treated of in another place (Hydro-Therapeutics). A warm bath, it may be added, reduces fever by the succeeding dilatation of the skin, blood-vessels, and increase of perspiration.

Increasing Excretion of Fluid.—All the drugs which increase the secretion of sweat or dilate surface arterioles facilitate heat-loss. Opium, for example, and to a less extent morphia, ipecacuanha, ammonii, and potassii acetas, nicotina, and antimonial salts act upon the sweat centres of the spinal cord; pilocarpina and alcohol probably influence the nerve-endings in the sweat-cells; while a number cause dilatation of the arterioles through the vaso-motor mechanism.

Alcohol.—One of the most potent antipyretics which we possess is the much-abused and decried substance alcohol. Little need be said about its powers. It is invaluable in influenza, useful in catarrh, and, as an evidence of its value, it may be added, one of the best antipyretics in the pyrexia of phthisis; absolute rest in bed, fresh air, and cognac constitute all the measures taken by the physicians in more than one sanatorium for pulmonary disease against pyrexia, and with striking success. Alcohol is supposed to act as an antipyretic through its mild diaphoretic and diuretic properties, and by its retardation of metabolic processes. It also most probably has some stimulating effect upon the nerve-centres, which may be transient, but can be renewed by small doses frequently repeated; inciting thereby a more vigorous supervision of body temperature on the part of the thermotaxic centre.

Secondary Antipyretics.—By this term all measures taken or drugs given to remove from the body any existing cause, generally local in character, are denoted. They are as truly antipyretic, strictly speaking, as the drugs which act through the nervous system. A purgative often lowers or aids in lowering a high temperature by removing the primary or the accessory causes of pyrexia. Any measure adopted for the relief of tension, in an abscess or elsewhere, are antipyretic.

Uses.—The invariable and routine exhibition of antipyretic drugs is not to be commended. The presence of fever is only a sign of the existence of a cause, and in many cases removal of the determining cause is better practice than forcible subdual of the fever, till what time the evil subsides or proves fatal. Where the temperature of the body has risen owing to the presence of poisonous substances in the blood, it is only reasonable to strive to remove them by inducing their expulsion; in the sweat, urine, or fæces, or if due to localised foci, to free them by the knife; not to endeavour to suppress nature's method of resistance by lowering the temperature through artificial and central means. On the other hand, some antipyretic drugs act on the causal agents of pyrexia as well as upon the heat-regulating mechanism. So fever in influenza, ague, and acute rheumatism appears to abate after the exhibition of special remedies by reason of their action on the poisons causing these diseases, as well as by their influence on the thermic mechanism.

As phenazonum (antipyrin) and many other members of the same group of antipyretic drugs act as depressants of the great nerve-centres in the medulla, it is imperative upon physicians prescribing them to thoroughly grasp the dangers which may accompany their use for the purpose of subduing feverish conditions; still more is it unwise to allow the public to make trial of them indiscriminately without advice.

It is well in considering the advisability of attempting to reduce the fever in any case to bear in mind that, unless the pyrexia is dangerously high and the causative agent itself not amenable to antipyretic drugs, one should seek to remove the cause rather than to forcibly subdue the fever. If the presence of fever apparently aggravates the condition of the patient, deal with it so as to cause the smallest amount of ill-effect and disturbance to the primary disease. Never give full doses of the synthetic antipyretic remedies to patients who have never taken them before, and if the heart's action be even slightly impaired, combine with them some cardiac stimulant.

Antiseptic Surgery. *See* Aseptic Treatment of Wounds.

Antispasmodics.—Antispasmodics (*ἀντί*, against; *σπάσμα*, a spasm) are remedies which relieve or prevent spasm. The term "spasm," as used in medicine, is applied to muscular contraction which is in some way excessive, useless, or injurious. Muscular spasm may be general or local; it may affect voluntary muscle, or involuntary; it may be "tonic" or "clonic" in character; it may be the result of a great variety of conditions, some of which are mentioned below.

Enumeration.—Antispasmodic remedies, accordingly, belong to many classes, and find their appropriate uses in quite different cases. Among the more important may be mentioned—opium, belladonna, stramonium, cannabis indica, conium, lobelia, alcohol, ether, chloroform, amyl-nitrite, nitrite of soda, and nitro-glycerine; the bromides, iodide of potassium, valerian, asafœtida, musk, castor, galbanum, camphor. Among adjuvants may be mentioned—warm baths, steam, cold sponging, friction, exercise, and certain tonic remedies such as arsenic, phosphorus, quinine, zinc.

Action.—Muscular spasm may be due to loss of control of the higher nerve-centres, whose function is to keep the lower (spinal) nerve-centres and the local neuro-muscular mechanisms in subordination (paralysis with spasm); or to irritative lesions of the higher motor centres (Jacksonian epilepsy); or to increased activity of the lower nerve-centres, either from local (strychnine poisoning) or reflex irritation (spasm associated with phimosis); or of the local neuro-muscular structures; or to general causes, whether psychic (hysterical spasm), toxic (uræmic convulsions), or nutritional (the spasmodic affections associated with rickets). Thus some antispasmodics act by stimulating the higher nerve-centres, as alcohol and ether; camphor stimulates the brain, the spinal cord, the circulation, and respiration; belladonna soothes the respiratory nerves, while stimulating the respiratory centre; conium paralyses the motor nerves; the bromides depress the motor (reflex) centres in the spinal cord; amyl-nitrite produces vascular dilatation by paralysing the muscular coat or the ends of the nerves. Valerian and asafœtida possibly owe their esteem in part to the volatile oil they contain, and in part to the mental effect produced by their disagreeable taste and odour.

Uses.—*Belladonna* is used in diseases of the respiratory organs for the relief of spasmodic asthma, spasmodic cough of all kinds, and especially of whooping-cough; in diseases of the genito-urinary organs such as chordee, the nocturnal incontinence of children, painful spasm of the bladder in calculus, cystitis, or prostatitis, and for renal colic; also locally for spasm of the sphincter ani. Stramonium is chiefly used for spasmodic diseases of the respiratory organs.

Chloroform may be administered in uræmic or puerperal convulsions, tetanus, the status epilepticus, severe chorea, renal or hepatic colic, and in spasmodic asthma.

The Nitrites are used chiefly for relaxing arterial spasm. Amyl-nitrite is used largely for angina pectoris associated with high tension; it may also be useful in some cases of infantile convulsions, and in epilepsy it may abort a fit coming on. It is sometimes useful in asthma.

Asafœtida, *valerian*, and their allies are used chiefly in hysterical conditions; also as enemata in intestinal colic with flatulence.

Iodide of Potassium produces great benefit in some cases of asthma, especially in those associated with emphysema and chronic bronchitis.

Antipyrine is useful in cases of pertussis, and has been recommended for epilepsy. The effect of heat is recognised in the popular use of a warm bath in infantile convulsions; of cold, in the beneficial effects of the cold douche in laryngismus stridulus.

Tonic and dietetic treatment has to be considered in most cases, as is strikingly shown by their effects in the treatment of the convulsive disorders associated with rickets—convulsions, head-nodding, laryngismus stridulus, and tetany.

Antitoxins. *See* MICRO-ORGANISMS.

Antrum. *See* NOSE (Accessory Cavities).

Anuria. *See* URINE.

Anus. *See* RECTUM.

Aorta (Thoracic), Aneurysm and Dilatation of.

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PATHOLOGY.—The existence of intrathoracic aneurysm probably always implies changes in the vascular coats preceding the local dilatation, which may be accidental in the sense of its being the result of some sudden effort made by the patient and consequent sudden strain upon the weakened aortic wall. The history of a severe blow over the upper part of the chest is not very rare in cases of aneurysm of the arch. In a case of abdominal aneurysm under the care of the writer, the patient, a man under thirty years of age, received a severe blow on the abdomen, and two years later came to hospital suffering as stated. A large aneurysm was found post-mortem associated with profound disease of the abdominal aorta, the rest of the arterial system being practically sound. Dr. Kelynack has related to the writer a case in which the aorta was cut across by the slipping down of a gas-meter upon the patient's abdomen, though there was no injury to any part of the intestine, and only a bruise on the anterior abdominal wall. The hæmorrhage was entirely retro-peritoneal. The relationship of habitual severe physical effort to aneurysm, again, seems to be well founded, and in a proportion of cases a part in the degenerative process that precedes the yielding of the vascular wall may with likelihood be attributed to syphilis. Especially is this so in the younger subjects of aneurysm who have been also subjected to physical strain. As might naturally be supposed, middle age is the usual period of development of aneurysm when the aorta tends to become degenerated by the atheromatous process, and the patient is still active. Exceptionally typical aneurysm arises at a comparatively early age, as in a case of the writer's, in which the patient was a man aged twenty-two, who had never had syphilis, and who had not been exposed to physical effort. The origin, thus, of a few cases is quite obscure, and some congenital imperfection of structure is suggested.

Clinically the division of aneurysms into classes according to their shape, structure of their coats, and the relation of the blood to these, etc., is of little or no importance, apart from the fact that some of the terms, as *true* and *false* aneurysm, have actually been used in opposite senses. For the clinician an aneurysm is *a special kind of tumour*, and its symptoms for the most part are those of pressure on surrounding structures. It has no auscultatory sign, moreover, that is not represented in simple dilatation of the arch, in which condition pressure symptoms are practically absent. The peculiarly destructive property of aneurysmal pressure, so well illustrated by its destruction of bone that may obstruct its expansion, is probably associated, not only with the high degree of pressure, but to some extent also with the incessant variation from maximum to minimum blood-pressure as the result of cardiac contraction.

It is not surprising, moreover, that living tissues should resent such pressure by generally undergoing inflammatory changes, and finally atrophy. This is well seen in the case of absorption of the bodies of the vertebræ before advancing aneurysm, while the more lowly organised and somewhat yielding intervertebral discs are well preserved.

The pathological process in the coats of the aorta itself has been referred to as degenerative, and so essentially it is, though an inflammatory process of low intensity is clearly indicated in the early stages. Not only does atheroma affect the aortic tissues in the degeneration period of life, but the products of the early inflammatory change almost from their inception are prone to degradation.

No attempt has been made here to deal with the process of aneurysm formation in general. For instance, the aneurysm that results in arteries—especially the cerebral—from embolism and the acute small aneurysm that may be produced in the aorta itself by micro-organisms in septic endocarditis cannot be included in this description. The writer has seen an aneurysm the size of a small nut produced by the latter process in the aorta close to the valves, and it is conceivable that, had the patient survived, this might have so enlarged as to give origin to the usual pressure symptoms and signs of ordinary aneurysm of the arch. The septic condition itself is, however, so inevitably fatal that no practical importance can attach to such a mode of origin of aneurysm.

The process of clot-formation, no doubt, may be regarded as an attempt at cure on the part of nature, but in the case of all but the smallest aneurysms of the arch, nothing more than the attempt can be discerned.

PHYSICAL SIGNS.—*Inspection.*—Unless the aneurysm be small or situated in the posterior portion of the arch, or in the descending thoracic aorta, it seldom happens that careful inspection of the chest in a good light fails to reveal some degree of *abnormal pulsation* distinct from that of the heart. Facing the patient and looking towards the front of his chest it is possible, however, to miss pulsation that is at once revealed by a side glance; and this may happen quite as easily in the case of large aneurysms as in that of those of moderate size. It is important, then, to look carefully across the front of the chest from the sides or downwards from over the patient's shoulders. Any local bulging there may be, should be carefully examined for pulsation, as it not rarely happens that a distinct projection or tumour has been formed by the aneurysm, and such tumour pulsates and is expansile. When the pulsation of the heart in its ordinary situation can be identified, the possibility of any visibly pulsating area being that of a displaced heart is disposed of. In "fibroid phthisis" of the left lung the heart, for instance, may be seen pulsating under the left clavicle, but then there is absence of pulsation from the normal cardiac area. Again, there are certain extremely rare conditions in which an effusion in the pleura or consolidation of lung on the left side may assume pulsation, no doubt derived from the adjoining heart, but the ordinary physical signs of such pleural and lung states will be obvious to prevent a careful observer from mistaking such pulsation for aneurysm. Lastly, in aneurysm of the descending aorta situated behind the heart a widely diffused pulsation, apparently synchronous with the pulsation of the heart, may be observed over the whole cardiac area, and to some extent it may seem beyond it. Under the circumstances it may theoretically be supposed that a double impulse would be observable, but if such there be, it is practically impossible of recognition. Moreover, a similar phenomenon may be met with in the case of a large new growth situated behind the heart, which seems to show that it is the pulsation of the heart itself that is conspicuous rather than that of the aneurysm. The visible pulsation produced by simple enlargement and hypertrophy of the heart retains more or less the physiological local impulses of the organ—the apex-beat and epigastric impulse—and when the right side of the heart is specially involved there may be visible impulse above the third cartilage on the left side, due to dilatation of the infundibulum of the right ventricle. But in the case of the heart being, as it were, thrust forward by pressure from behind, the whole organ seems to be projected forwards. It must be remembered that the heart in diastole is like a flaccid sac in comparison with its state in systole, which is that of a mass of rigid muscle. In systole the heart so asserts itself that surrounding parts must yield before it, and in the case of aneurysm or neoplasm encroaching upon its space from behind it still so behaves.

Large aneurysms of the descending aorta may produce a pulsating area behind to the left of the spinal column.

Any *asymmetry in the shape of the chest* should be noted, for an aneurysm often compresses a main bronchus and so interferes with the play of the lung—generally the left—or it may induce pleural effusion or secondary consolidation of the lung with purulent infiltration. This last result is more often seen in cases of solid growth within the chest.

Exaggerated arterial pulsation in the neck (and elsewhere) is seen when the aortic valves leak, as a result of aneurysm involving the first part of the arch. A difference between the carotids is common without aortic incompetence, but in the presence of this lesion such difference is rendered very striking. Again, the *superficial veins* of the neck are often seen to stand out, motionless and knotted at the sites of the valves, when there is pressure of the aneurysm upon the superior cava or innominate veins, offering a contrast to the same veins as they are seen to pulsate in ordinary heart cases. The obstruction in the large venous trunks produced by aneurysm of the arch in its transverse portion leads sometimes to a remarkable appearance owing to the engorgement of the superficial veins of the front of the chest, arms, and head and neck. In the first situation the enlarged and tortuous veins may resemble a black breast-plate, so thickly are they set. Finally the seats of venous engorgement tend to become œdematous. This condition is often spoken of as if it were peculiar to solid growth within the chest, but it occurs in aneurysm by no means very rarely. An aneurysm acts in precisely the same way as such a growth—it is in fact a tumour. The abdominal veins may show some fulness, which is rendered very evident when the patient coughs. Quite apart from aneurysm, and in healthy individuals, a double arch of fine venous varicosity is often seen along the double costal arch. This arch of venules marks the separation of the venous current upwards and downwards to the superior cava and inferior cava respectively. When there is obstruction superiorly there will be excess of blood passing downwards, and hence the fulness of the abdominal superficial veins. In a case of the writer's a fulness of the abdominal veins became marked, while the thoracic engorgement, which at one time had been intense, diminished.

"*Tracheal tugging,*" to be described under palpation, is occasionally quite distinct on inspection when the patient raises his chin.

Palpation.—If an aneurysm projects beyond the general surface of the chest it will certainly be *seen* to be the seat of pulsation, but only the hand can convey the impression afforded by the powerful heave of the sac, which feels well-nigh irresistible in force so long as the heart remains vigorous. It is only too well known how such force, remittingly applied, can slowly destroy the hardest tissues composing the body that oppose its advance, and the agony the process implies to the patient is only too often experienced. Usually the seat of palpable pulsation is the front of the chest, and it is only rarely that an aneurysmal sac reaches the posterior aspect, bulging or corroding the ribs, but it quite commonly corrodes the bodies of the vertebræ, although we may fail to feel pulsation either back or front. Aneurysm is thus one of the things that obstinate pain in the back-bone should always suggest. These latter remarks of course apply to aneurysms of the posterior portion of the arch or of the descending aorta. Towards the front of the chest the sternum cartilages and ribs offer feeble and therefore less painful resistance to the extension of the sac. The moment a tumour is produced a most valuable character pertains to it, namely, *expansile impulse*. When such a tumour is grasped between the fingers and thumb, these are felt to be separated with each systole of the heart. Sometimes a systolic *thrill* or *thrills*, systolic and diastolic, or almost continuous through both periods, can be felt over prominent aneurysmal sacs. Usually when there is a diastolic or double thrill the aortic valves have become incompetent, but the writer believes not always, at least he has felt such a continuous thrill when the incompetence of the valves was difficult to demonstrate post mortem. In aneurysms of the ascending and transverse portions of the arch, a more common palpation sign than thrill is the *peculiar shock* indicative of and coincident with the closure of the semilunar valves with extraordinary force.

Palpation of course is employed to determine any difference there may be between the right and left carotids, radials, etc., to ascertain the position of the cardiac impulse, and to compare the force of this latter with the force of the aneurysmal impulse. The force of the latter being at least as great is a valuable sign of aneurysm.

The aneurysmal sign that came recently into vogue—"*tracheal tugging*"—is elicited by palpation of a special kind, which consists in gently supporting the lower border of the cricoid cartilage on each side with the forefinger and thumb, the patient's chin being elevated; presumably this sign is best developed in cases of aneurysm involving the transverse portion of the arch, but when it is present there is usually abundance of other evidence in favour of aneurysm. As already mentioned, "tracheal tugging" may be an inspection as well as a palpation sign.

Percussion.—The dull area produced by aneurysm of the arch in the front of the chest at its upper part is characterised by a very definite distribution: it crosses the median line and it reaches neither costo-acromial angle. It is thereby at once distinguishable from lung dulness. In the immense majority of aneurysms of the arch this rule holds good, but as an instance of the exception that proves the rule, a case may be mentioned which was equally applicable for narration under inspection and palpation, and which illustrates how seemingly very odd occurrences may have simple explanations. A patient had a large pulsating dull area in the right mammary region under the right nipple, with its maximum intensity some four inches from the median line. Post-mortem examination revealed a large pear-shaped aneurysm coming off almost at right angles by a narrow neck from the right side of the first part of the arch. Such an aneurysm could not occasion the dull area described above as characteristic of this disease in the arch. In thus referring to exceptions to rules opportunity may be taken to remind the reader that apparently anomalous aneurysmal signs may depend upon the fact of more than one aneurysm being present. To return to the ordinary distribution of dulness in aneurysm of the arch. The degree of extension to right and left respectively varies much, and in some cases the middle line is little more than distinctly crossed. As a whole, the dull area assumes a somewhat semilunar shape, and is usually separated from the area of cardiac dulness, but there may be a more or less narrow "neck" of dulness uniting them. It is obvious that the size of the aneurysm and the size of the dull area produced by it will not correspond, inasmuch as the dull area only indicates the extent of the surface of the sac in contact with the chest wall, the lung having been displaced, and does not indicate the diameter of the sac. Very rarely tubercular consolidation of the apex of one or other lung complicates aneurysm, and then there may be a combination of the "mediastinal" dulness with the dulness of the pulmonary consolidation. The dulness of consolidation of the lungs dependent directly on aneurysm is usually found at their lower part—at all events at first—and the same may be said of pleural effusion. In a case of intrathoracic tumour the writer once found the lower lobe of the left lung consolidated and infiltrated with pus, while the upper lobe was compressed by pleuritic effusion, evidently of later date, the consolidation having been produced first of all as the result of pressure on and obstruction of the main bronchus of the lower lobe. A similar condition might be met with in aneurysm.

Auscultation.—It will usually happen that in a methodical physical examination of the chest the signs obtained by the other methods of physical diagnosis have established the diagnosis of aneurysm before auscultation is reached. Not the less, however, is auscultation necessary, for it puts us in possession of facts of immense importance in confirmation of the diagnosis. The moment the stethoscope is applied to the aneurysmal surface the auscultator is often aware of an *impulse or jog*, while bystanders see his head moving synchronously with the patient's cardiac systole. The first sound is often absent over the sac, and is by no means necessarily replaced by a murmur, all that the auscultator is aware of being the jog or impulse just referred to, which is conveyed to his cerebral centres by the nerves of common rather than auditory sensation. There may, however, be a more or less loud *systolic murmur*, with or without a systolic sound.

It is the second cardiac sound that, in aneurysm involving the first or first and second parts of the arch, bears such characters as are practically pathognomonic. Great *intensification* or *accentuation* of this is the sign referred to. So forcibly are the valves closed that the shock of their closure may in many cases be easily felt following the systolic expansion or heave (*vide* "Palpation"). This intensification of the aortic second sound is a sign of peculiar value, and if a practised observer has neglected systematic physical examination and employed auscultation first of all, he will in most instances be at once put in possession of the diagnosis. It is very common for such a second sound to be followed by a *murmur*, of course *diastolic in rhythm*, and indicative of some leakage taking place between the overstretched valves, which are unable to close completely the enlarged orifice. It is possible, however, that a diastolic murmur may be produced in an aneurysmal sac apart from incompetence of the aortic valves. At least in cases of the kind it may be impossible to demonstrate post-mortem such a degree of incompetence as would explain so loud a murmur as that heard, but it must be remembered how impossible it is post-mortem to represent the state of the valves in the presence of high blood-pressure during the patient's life. Over some large aneurysms a *practically continuous murmur* may be heard running through both systole and diastole, and in cases of the kind incompetence of the aortic valves may not be easy of demonstration post-mortem. On the other hand, incompetence of the aortic valves may be abundantly evidenced post-mortem, not only by the state of the orifice and its valves, but by the condition of the left ventricle, which may be considerably dilated, while the murmur of mitral regurgitation was audible towards the end of the patient's life.

The accentuation of the second sound heard over aneurysm of the arch is usually of much greater intensity than that characteristic of mere dilatation of the vessel, though the latter sound is often of great loudness, and may be clear, ringing, and almost musical. The aortic second sound in true aneurysm, however, is indicative of the closure of the valves with a degree of force that seems almost irresistible, and that is never reached in simple dilatation of the arch. In simple dilatation pressure symptoms, moreover, are absent.

SYMPTOMS.—*Pain* is an exceedingly common symptom of intrathoracic aneurysm. It varies in intensity, and in aneurysm of the arch is often referred to the upper part of the front of the chest, shooting to the shoulders, and sometimes into the neck. In aneurysm of the descending aorta, again, pain in the back is apt to be of great intensity and persistence, owing to the pressure exerted on the spinal column. Lastly, in aneurysm involving the first part of the arch, true angina pectoris may be produced by implication and consequent narrowing of the coronary orifices by the atheromatous process, though this event is more common in cases of simple dilatation of the arch than of true saccular aneurysm. When true angina occurs, the radiation of the pain is the usual one, down the ulnar side of the left arm and forearm to the ends of the little and ring fingers. Moreover, the pain may be followed by the numbness and powerlessness of the classical angina. But irregular forms of angina are perhaps more common. In certain cases of aneurysm of the arch, probably involving the innominate artery, pain and paresis are experienced in the right arm. Then there is a curious localisation of pain that is most difficult of explanation, namely, in the occipital region. This pain is sometimes referred to one side, and may be of great intensity, so that it forms the most prominent symptom of the case.

General Weakness.—Some degree of general weakness and unfitness for exertion is usually a feature of the condition, but this symptom, like many of the others, shows great variations in its severity.

Respiratory Symptoms.—There is often pressure of the sac on the left recurrent laryngeal nerve as it courses round the aortic arch on its way to the larynx. At first the symptoms are those of irritation of the nerve with spasm, and later come paresis and paralysis. Patients with aneurysm of the arch often have a very characteristic ringing—so-called *"brassy"—cough.* Such a cough at once suggests aneurysm to the practised observer. The cough is dry, and there are at this stage no indications of catarrh or other laryngeal or bronchial cause for it. Later, catarrhal conditions of the bronchi are common. When the left cord is palsied the cord is seen, on laryngoscopic examination, to be motionless in the cadaveric position, while the larynx is normal in colour and structure. The writer is convinced that the palsy may remit or intermit before becoming permanently established. The voice may not be much altered owing to the moving cord being still able to meet its fellow. There is usually, however, some degree of hoarseness, and there may be a degree of inspiratory stridor. Before the laryngoscope came into use these symptoms were, naturally enough, attributed to inflammation of the larynx, and the patient was subjected to local treatment that was altogether useless, and often produced suffering.

Several cases are on record in which bilateral paralysis of the cords has resulted apparently from pressure on one vagus—not recurrent—nerve. The only explanation that seems at all feasible is that the nerve nucleus associated with the nerve has itself become affected in a secondary manner.

This granted, we know how closely associated are the nuclei presiding over the vagi. For instance, the vocal cords always act together physiologically. The right vocal cord may be paralysed in a case of intrathoracic aneurysm, owing to involvement of the subclavian artery in aneurysmal disease, as the writer has seen. The possibility of there being two or more aneurysms must never be forgotten in the explanation of complex symptoms.

Laryngeal paralysis may constitute almost the only indication of intrathoracic aneurysm. In one case seen by the writer this was so. The patient had a slight though obstinate bronchial catarrh, but otherwise seemed in good health. Fortunately his larynx was examined, though his voice seemed normal, and a palsied left cord found. A few weeks later the patient dropped down and died in the street, no doubt from ruptured aneurysm. It must not be concluded at once, however, that a patient with a palsied left cord and an otherwise normal larynx is suffering from aneurysm. The recurrent nerve may be compressed by tumours or enlarged lymphatic glands, or may be caught in the changes associated with fibroid phthisis; instances of the last two accidents have come under the writer's notice. When inflammatory changes are present in the larynx, palsy of one or both cords goes for little in diagnosis; the cause may be only the contiguous inflammation. The laryngoscope may afford invaluable aid in diagnosis, even by showing that the larynx is normal in its motion as well as in colour and structure. When there is manifest respiratory—generally inspiratory and expiratory—stridor and the larynx is normal, the pressure of the aneurysm must be exerted on the trachea and main bronchi, and is thus direct. The recognition of the seat and nature of the obstruction in such a case is necessarily a matter of supreme importance when there is question of tracheotomy. It has unfortunately happened on more than one occasion that laryngotomy has been performed when the obstruction was not indirect and due to spasm of the glottis, but direct and due to pressure of the sac on the trachea lower down. There may be some *deviation of the trachea*, and the physical sign already described, tracheal tugging, will usually be pronounced. The stridor is both inspiratory and expiratory, and the voice may be feeble, apparently from the deficient current of air passing the cords. The general resemblance of the symptoms to those of obstruction situated in the larynx is considerable, and a laryngoscopic examination may be the only means of ascertaining, by exclusion, the seat of obstruction.

Bronchitis generally supervenes in cases of aneurysm of the arch compressing the trachea or bronchi, and suffocative paroxysms are apt to be induced by muco-purulent accommodation blocking up the narrowed channel, while *paroxysms resembling asthma* frequently occur, possibly having their origin in spasmodic contraction of the muscular fibres of the bronchi. Some months ago the writer had under his care a case of aneurysm of the arch in which he could not find any laryngeal paralysis, although before admission to the infirmary the patient described suffocative attacks of such violence that during them he fell down partially unconscious. The patient came recently under his observation, and there was still no laryngeal paralysis, while indications of pressure upon the lower part of the trachea and left bronchus, previously present, had become pronounced. When there is pressure of an aneurysmal sac on the trachea the expansion of the lower zone of the chest is interfered with, and inspiratory retraction of the same is common as in other obstructions of the air-passages. Secretion collects in the bronchi, and both inspiratory and expiratory movements become laboured efforts. The patient sits up in bed and leans forward as if to relieve the pressure. This attitude may thus become suggestive of the disease. The same attitude is, however, observed in stenosis of the trachea from syphilis. When the larynx is the seat of obstruction its "respiratory excursion" becomes increased, and the head is generally thrown back. When one bronchus, usually the left, is compressed, certain unilateral signs result. There is deficient expansion, and there may be a slight change in the percussion resonance on the affected side, while the respiratory murmur is simply weak or replaced by rhonchi. When unilateral, such rhonchi are significant of pressure, ordinary bronchitis being a bilateral condition, but pressure on the trachea, as well as on a bronchus, is common. A bronchus may become completely obstructed, and then the lung may pass into a pneumonic condition, often with purulent infiltration, such as is more commonly seen in cases of intrathoracic new growth.

Rises of temperature, to a considerable height, but of short duration, are often witnessed in cases of aneurysm under treatment without there being evidence of obvious cause. Possibly the iodide treatment has had something to do with their occurrence. The pulse-rate is often increased in cases of intrathoracic aneurysm, and such increase is probably promoted by large doses of iodide of potassium: the subject will be referred to later.

The phrenic nerves are said to be involved in rare cases of intrathoracic aneurysm; the patients suffering from spasmodic contraction of the diaphragm with a sensation of constriction round the base of the chest. The writer has had no experience of the condition.

The pupils and palpebral fissures are occasionally rendered unequal by interference with sympathetic fibres, the left side being the affected one. Irritation of these fibres induces enlargement of both pupil and fissure, paralysis — the more common — diminution of both pupil and fissure. Unilateral disturbance of the secretion of sweat may occur, but neither of these conditions is peculiar to aneurysm.

Dysphagia may result from direct pressure of an aneurysm on the gullet. Usually the degree of pressure is not extreme, and like most aneurysmal symptoms the dysphagia is curiously, and seemingly capriciously, liable to vary in severity. In a few cases some degree of *emaciation* is present, due to interference with nutrition from compression of the thoracic duct.

General anasarca can hardly be regarded as a feature of intrathoracic aneurysm, but in aneurysm involving the first part and inducing aortic incompetence, it may be present as in ordinary heart cases. The special dropsy that occurs as the result of pressure by the aneurysm on the superior cava and innominate veins has already been considered under "inspection" in the section devoted to physical examination of the chest.

Arterial or pulse changes, as far as the carotids are concerned, have been included in the same section, but it is as impracticable as undesirable to make an absolute distinction between physical signs and symptoms.

The Pulse.—It must be remembered that without referring to anatomical irregularity of the arteries, aneurysm is not the only disease that may alter the radial pulse of one side. Local disease of the origin of subclavian or innominate arteries, quite apart from aneurysm, may diminish or annul one radial pulse. Moreover, in very rare cases of intrathoracic aneurysm both radial *pulses* may be annulled without any interference with the nutrition of the parts supplied being noticeable. Local atheromatous disease—apart from aneurysm—at the origin of the innominate artery will interfere with both carotid and subclavian trunks of the right side, while on the left side local disease may produce a similar result by implicating both orifices in the aorta. Setting aside such fallacies, aneurysms interfere with radial pulses in different ways. The classical explanation given is the interposition of the elastic sac between the heart and affected vessel, but it is obvious that there are other ways in which an aneurysm may interfere with the radial pulse of one side, as for instance pressure of the sac on the outside of the innominate or left subclavian trunks, or the origin of the main vessel being from the sac itself, in which case the orifice may be transformed into a mere slit, or be obstructed by fibrin deposited on the sac wall.

Without considering further the possible ways in which an aneurysm may interfere with a radial pulse, the characters of the radial pulse when so interfered with have to be studied sphygmographically. "There is alteration in the character of the pulse-wave" itself, and something more than a diminution in the diameter of the vessel and in the strength of the beat. A recent author remarks, "The most significant indication of aneurysmal interference with the pulse is delay of the beat." It is not the beginning of the pulse-wave that is delayed, however, but the reaching of its maximum intensity. The sphygmogram shows the upstroke to be more sloping, the height less, the summit of the curve more rounded and the down-stroke more gradual. So to palpation the pulse feels smaller, more compressible, and more continuously full between the beats. The pulse is more or less "smoothed down," to borrow a phrase from the author referred to (Sir Wm. Broadbent).

The late Dr. Fagge, in his *Practice of Medicine*, states that "if the aorta were converted into a large sac the impulse of the heart would be lost in this space, and the blood would flow out through the efferent vessels in a continuous stream." He states, "A case once in Guy's Hospital exemplified this, the pulse at the wrists became quite imperceptible, but the warmth of the hands and their vascularity showed that the blood flowed into them as before." The writer made a similar observation some years ago, and can verify Dr. Fagge's statement from his own experience.

Course and Terminations.—Few diseases justify a more gloomy prognosis than intra-thoracic aneurysm, and yet the disease may be survived for years, and this, too, without any great measure of suffering. What may be termed the accidents of the disease are numerous, and for the most part speedily lethal. First of all, the sac may rupture and effusion of blood may take place externally through the skin, when it is to a certain extent under control. The skin, however, may become red or bruise-stained, and yet the threatening process be stayed. More frequently hæmorrhage takes place into a serous potential sac, as the pleura or pericardium, or into a mucous canal, as the trachea or œsophagus. In the latter cases the blood, of course, appears externally as a profuse hæmoptysis or hæmatemesis. Instead of enumerating in detail the parts into which intra-thoracic aneurysms have been known to rupture, the statement of the late Dr. Walshe, in his classic work, may be quoted as sufficiently comprehensive: "There is no conceivable position into which fatal rupture has not occurred," and mention may be made of three of the most remarkable, namely, the pulmonary artery, the vena cava, and the cavities of the heart itself. Even profuse external hæmorrhage from an aneurysm is not necessarily immediately fatal, and patients may live for days with a gradually increasing extent of chest wall, and aorta replaced by lint, in which state a patient of Dr. Walshe's lived for nearly two months. The late Dr. Ramsay, of Broughty Ferry, again, related the following extraordinary experience of a patient of his:—The man was the subject of a large projecting aneurysm of the arch, which one day ruptured externally, a stream of blood issuing from it. Nothing daunted, he coolly held a vessel to receive the blood till faintness came on, when the bleeding stopped. The patient recovered from this accident and lived several months, dying ultimately, not from his aneurysm, but from typhus fever! Even rupture into the gullet may be survived for several hours. In a case of this kind seen by the writer, acute delirium supervened during the time survived. In tracheal or bronchial perforation, oozing of blood, causing blood-tinged sputa, for several weeks may precede the final gush from rupture. In true internal rupture, as into the pleura, sudden pallor and convulsions from cerebral anæmia are the common symptoms.

An aneurysm of the posterior part of the arch, or of the descending aorta, occasionally reaches the spinal canal by corrosion of the vertebræ. The writer once witnessed the symptoms of this occurrence. The patient was under treatment in hospital, and suddenly cried out with intense pain in his back, which was only relieved by large hypodermic doses of morphia. Soon after the onset of the pain he began to complain of being unable to move his legs, and next morning was quite paraplegic. The diagnosis was verified by post-mortem a few days later.

But many intra-thoracic aneurysms prove fatal without the occurrence of rupture. The pressure of the sac upon the trachea and bronchi and the lung-changes that such pressure is apt to induce, are frequent causes of death; patients often die apparently from exhaustion and asthenia, but generally there is some acute terminal condition, such as pneumonia, while rupture of the sac internally is not rarely the cause of death, although it escaped clinical recognition.

TREATMENT.—It has been said with grim truth that the efficacy of a treatment bears inverse proportion to the number of methods and means employed. The treatment of intra-thoracic aneurysm offers no contradiction of the statement. Unquestionably, a great deal can be done to mitigate the sufferings of the patient, and when the disease is discovered early, to slow and possibly to stay its advance. Nature in the rarest of cases brings about practical obliteration of a small sac by clot-formation, but such small sacs have usually never given rise to symptoms calling for medical treatment. Of the numerous methods of local treatment employed with the view of originating and promoting clot-formation, it is impossible to give even a short description that would enable a reader to apply the treatment without reference to minute detail only to be obtained in original monographs. The writer has witnessed the application of one of these methods—that by galvanism and acupuncture—on several occasions, but he cannot say that he has seen results that make him in any way desirous of adopting the treatment. On the other hand, he has seen life prolonged free from any great suffering for a considerable period under the most unfavourable circumstances, as when a large portion of the sac wall has been composed of lint soaked in blood-clotting solutions, as in Dr. Walshe's case already related. He doubts if much better results have followed any of the ingenious attempts at the local treatment of intra-thoracic aneurysm that have been made. Such methods as entail pain and suffering to the patient are to be most strongly deprecated.

A great deal can always be done to relieve suffering by the hypodermic employment of morphia guarded by an infinitesimal quantity of atropine,—and this, too, when the relief seems hardly to be expected, as, for instance, when the sac compresses the trachea and produces paroxysmal dyspnœa. The writer has under such circumstances, however, been able rapidly to remove what seemed to be a suffocative paroxysm, and to repeat the treatment with an equally good result. On the other hand, when, owing to pressure of the sac on the trachea or bronchi, secretion has accumulated in the tubes behind the obstruction, this treatment is fraught with danger, unless care is taken to begin with what may seem absurdly small doses, although assisted by cyanosis these may accomplish the object for which they are given. In the treatment of pain due to pressure on the bodies of the vertebræ, on the other hand, morphia may be fearlessly "pushed" to the accomplishment of its object—the relief or removal of pain. There need be no apprehension in such cases of creating an "opium-eater" or morphio-maniac, provided the diagnosis be secure. There is no reason, either, to consider the "individual capacity for pain."

The ordinary treatment of sufferers from intra-thoracic aneurysm implies a prolonged rest in the recumbent posture—promoting "physiological rest" as far as such is possible to the circulatory apparatus. It is impossible to lay down the duration of such period of recumbency in individual cases—usually six to eight weeks suffice, the patient being *gradually* allowed to get up and move about. Harassing restrictions of fluid and food the writer deprecates; at the same time, common sense dictates abstinence from stimulants and stimulating food, and the taking of no more food and drink than are necessary to remove the pangs of hunger and thirst. Good nursing, in which is included the management of the bowels, and of the acts of micturition and defæcation, as well as the "making of the bed," is of supreme importance, and even in the houses of the poor, thanks to District Nurses, the friends of the patient can now receive efficient practical instruction in matters of the kind.

Of therapeutic means—other than for the relief of pain—the writer will refer to only one, the iodide of potassium, which he has employed for the last twenty-six years in the treatment of aneurysm, and in which his faith has somewhat waned. He knows, however, no better drug, nor any so good, and is by no means disposed to deny that its effect "makes for betterness." Long ago he was suspicious of the frequent acceleration of the pulse in cases of aneurysm being associated with the iodide treatment, and he was much interested in the more recent avowal of this belief by Dr. G. W. Balfour, to whom medical men generally, and aneurysm patients specially, owe such a debt of gratitude for vigorous therapeutic efforts on their behalf. The rules laid down by Dr. Balfour are to ascertain the "normal" rate of the patient's pulse before beginning the iodide treatment, to begin with moderate doses (10 grs.), and to be guided by their effect on the pulse-rate as to their increase. He tells us that "it is only rarely that we can increase the dose to 15 grains (every eight hours) without raising the pulse-rate."

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Aphasia.

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Definition.—In my opinion the term aphasia should be limited to those derangements of speech which result from functional disturbance or organic disease of the *higher cerebral speech-mechanisms properly so-called.* And by speech I mean *vocal speech or its direct equivalents* (*e.g.*, the written speech of educated people who are possessed of all their faculties, the tactile speech of blind persons, and the finger-speech and lip-reading of deaf persons). The term aphasia should not, in my opinion, include: the defects in the perception of spoken and written speech, which are due to disease in the end-organs of special sense—the ear and the eye; mere defects of intelligence, defects in pantomime, nor defects of articulation, such as are due to lesions in the lower cerebral speech mechanisms (the pons Varolii and medulla oblongata).

VARIETIES OF APHASIA. — The higher cerebral mechanisms concerned with speech are of two kinds, viz. *receptive* and *emissive;* consequently, the derangements of speech included under the term aphasia may be divided into two great primary divisions, namely, *sensory* aphasia and *motor* aphasia. The two chief forms of sensory aphasia are—(1) *word-deafness,* and (2) *word-blindness;* and the two chief forms of motor aphasia which are usually described are—(1) *motor-vocal aphasia,* and (2) *motor-writing aphasia* (agraphia); but many authorities now think that in many cases in which agraphia is present, the agraphia is not, strictly speaking, a true form of aphasia. With this opinion I entirely agree.

Further, since each of these varieties of aphasia may be due to lesions of — (*a*) the *speech centres themselves,* or (*b*) the *conducting tracts,* which pass to the sensory speech centres, or from the motor speech centre, two sub-varieties or forms of word-deafness, of word-blindness, of motor-vocal aphasia and of agraphia may occur, viz. subcortical and cortical word-deafness, subcortical and cortical word-blindness, subcortical and cortical motor-vocal aphasia, subcortical and cortical agraphia. The clinical differences between these different varieties will be afterwards described.

ANATOMICAL INTRODUCTION

THE POSITION OF THE SPEECH CENTRES.—The *auditory speech centre,* ASC, is situated in the posterior part of the first, and probably also of the second, temporo-sphenoidal convolutions. The *motor-vocal speech centre,* MSC, is situated in the posterior end of the inferior frontal convolution (the foot and ascending portions of Broca's convolution). The *visual speech centre,* VSC, is situated in the angular gyrus, and perhaps the adjacent part of the supra-marginal convolution. The exact position of the *motor-writing centre,* MWC, has not yet been definitely determined. Exner has suggested that it is situated in the posterior end of the second frontal convolution. In my opinion the motor-writing centre is probably merely a part of the ordinary psycho-motor centre for the upper extremity (hand and forearm), and is probably, therefore, situated in the ascending frontal or ascending parietal convolutions.

ASC = the auditory speech centre, to which external stimuli pass from the ear, probably through a lower auditory centre (not represented on the diagram).

VMSC = the motor-vocal speech centre, the action of which is (probably) chiefly controlled and stimulated by the auditory speech centre, and from which outgoing impulses proceed, the ultimate effect is to produce the muscular movements necessary for vocal speech.

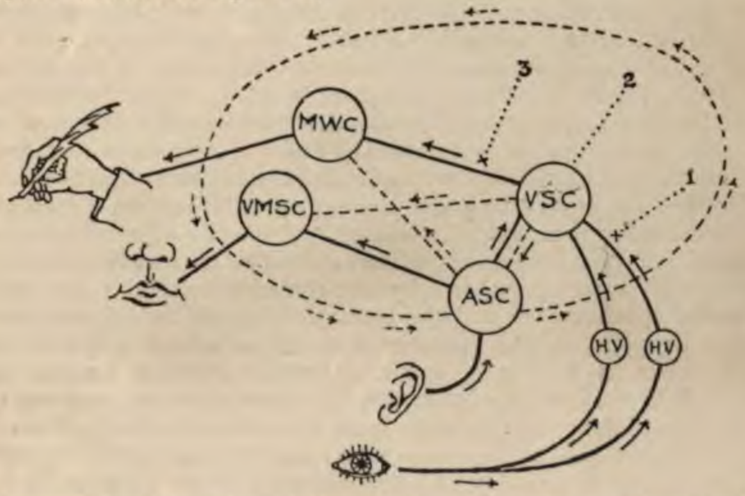
VSC = the visual speech centre, to which external stimuli pass from the eyes through the lower or half vision centres (HV, HV).

MWC = the motor-writing centre (probably not a *speech* centre), which is chiefly controlled and stimulated by the visual speech centre, and from which out-going impulses proceed, the ultimate effect of which is to produce the muscular movements concerned in writing.

Note.—Some authorities think that impulses from the visual speech centre pass through the motor vocal speech centre in order to reach the writing centre. In the diagram they are represented as passing directly from the VSC to the MWC; this, in my opinion, is their more probable course.

A lesion at 1 which destroyed the conducting fibres passing from *one* half vision centre to the visual speech centre would not produce word-blindness.

A lesion at 1 which destroyed the conducting fibres passing from *both* half-visual centres to the visual speech centre would produce word-blindness, but would not produce agraphia (the visual speech centre being unaffected and able to play upon the motor-writing centre).



A lesion at 2 which destroyed the visual speech centre would produce word-blindness and agraphia.

A lesion at 3 would produce agraphia (unless the motor-writing centre could be put into action in some round-about way), but would not produce word-blindness (the visual speech centre being intact).

THE RELATIVE ACTIVITY OF THE CORRESPONDING SPEECH CENTRES IN THE TWO HEMISPHERES OF THE BRAIN.—In the great majority of persons the *active* speech centres are situated on the side of the brain which is *for them*, the "active," "driving," or "leading" side. And since the great majority of persons are right-handed, the *active* speech centres are in most persons located in the *left* hemisphere of the brain. In left-handed persons, on the contrary, the active speech centres are usually located in the right hemisphere of the brain. Hence in right-handed persons an attack of right hemiplegia is apt to be associated with aphasia, while in left-handed persons the reverse is the case. Exceptions to this statement are, however, occasionally met with.

But although the active or driving speech centres are situated in right-handed persons on the left, and in left-handed persons on the right side of the brain, there is, I think, every reason to suppose that the corresponding centres in the opposite (non-leading or non-driving) hemisphere are possessed of some sort of speech function. Further, I believe that the relative activity of the speech centres in the inactive (non-leading) hemisphere may not only vary in different individuals, but that it varies, in some degree, with the special function of the speech centre concerned; the bilateral representation is probably most marked in the case of the auditory speech centre; but probably also obtains, in some degree, in the case of the visual speech centre and the motor-vocal speech centre. We may say, then, that the speech centres are bilaterally located; but that, in the great majority of persons at all events, they are only sufficiently educated to actively and independently carry on the speech function on one side of the brain (*i.e.* the side of the brain which in the particular individual is the "driving" side). Exceptions to this statement do, however, occasionally, though rarely, occur.

The existence of two centres for each speech faculty (in the right and left hemispheres of the brain), the functional activity of which may perhaps vary in some, though usually only in a slight degree in different individuals, and with the special speech faculty involved, is probably one explanation of the somewhat puzzling differences in the clinical phenomena which are met with in different cases of aphasia, and especially of the differences which occur at different stages of the same case.

THE BLOOD-SUPPLY OF THE SPEECH AREA.—The middle cerebral artery supplies the speech centres as well as the motor area of the cortex, and it is important to remember that the speech centres are situated on the outskirts of the area of its distribution, and that in some brains the speech centres may receive a supplementary blood-supply from the adjacent arteries; in other words, in some brains the auditory speech centre and the visual speech centre seem to be partly supplied by the posterior cerebral artery, and the motor-vocal speech centre and the motor-writing centre (if it is situated, as Exner supposes, in the posterior end of the second left frontal convolution) seem to be partly supplied by the anterior cerebral artery. Hence, embolic obstruction of the nutrient artery which supplies any one of these speech centres will, in most cases (*i.e.* those in which there is no supplementary blood-supply), produce complete and persisting aphasia, which in the great majority of cases can only be recovered from by the education of the corresponding centre in the right hemisphere; whereas, in a small proportion of cases (*i.e.* those in which there is a supplementary or inosculatory blood-supply) it will merely produce a temporary aphasia.

PHYSIOLOGICAL INTRODUCTION

THE NATURE OF SPEECH.—The act of speaking consists in the production of a series of highly elaborate co-ordinate muscular movements, the outcome of which is the production of certain sound vibrations (spoken words) to which a definite meaning is attached. A spoken word may be regarded as the motor equivalent of the sensory impressions which are produced in the auditory centre by the sound vibrations of which that word is composed; and the idea or memory of a spoken word, or rather of the muscular movements which are necessary for its production, consists in the revival in consciousness of the sensory impressions which attend the production of that movement. In the case of a spoken word these sensory impressions are chiefly auditory, partly visual and kinæsthetic. The idea or memory of a written word consists in the revival in consciousness of the sensory impressions which attend the act of reading and writing. These memories are chiefly visual, partly kinæsthetic.

APHASIA DUE TO A PROCESS OF DISSOLUTION. — The derangements of speech and intellectual language included under the term aphasia are degradations of function produced by disease; in other words, they are the results of a process of *dissolution;* whereas the development and training of the speech mechanisms and speech functions are the result of a process of *evolution*. In typical cases of total aphasia the patient is reduced, so far as his speech functions are concerned, to the position of a child who has not yet acquired the power of understanding spoken language, of speaking, of reading, and of writing. But in many cases of aphasia the process of dissolution is incomplete. The best way to obtain an insight into the pathological physiology of aphasia is to contrast the speech derangements which result from disease with the different stages of speech development in the young child. I cannot insist too strongly upon this point. It is, in my opinion, the very basis of an intelligent comprehension of the whole subject of aphasia.

THE EVOLUTION OF THE SPEECH FACULTIES.—In the development of the speech faculties in the young child, the auditory speech centre is first opened up, and it is only after it has reached a certain, though very rudimentary, degree of education that the associated motor-vocal speech centre begins to become really active as an intellectual machine. The infant, long before it can talk, can comprehend much that is said to it. In the course of time, but only as the result of much practice and repeated efforts, the action of the motor-vocal speech centre becomes perfected. In this process of education the child first learns to connect certain sounds and combinations of sounds (names and words) with certain objects, actions, attributes, etc., and to repeat many words like a parrot (*i.e.* without understanding their meaning); then to answer questions, and finally to talk spontaneously. The auditory speech centre and the associated motor (vocal) speech centre —the *fundamental* speech centres—acquire a high degree of functional activity in every normal individual, however uneducated he may be. Hence word-deafness and motor-vocal aphasia are met with in the most uneducated persons. It is only long afterwards that the visual speech centre and the associated motor centre (the motor-writing centre) are trained to play their part as active speech mechanisms; and even nowadays these *accessory* speech mechanisms, as they may be termed, are sometimes untrained, for one still meets with illiterates who are unable either to read or write.

PATHOLOGICAL INTRODUCTION

The sensory aphasias (word-deafness and word-blindness) may be regarded as *sensory paralysis;* motor aphasia may be regarded as a *motor paralysis;* and paragraphia may, I think, be regarded as an ataxia of speech, exactly comparable to the ataxia of tabes dorsalis, and like it associated with more or less marked sensory speech defects, and due to a lesion on the ingoing or sensory side of the cerebral speech mechanism.

CAUSES OF APHASIA.—In some cases of aphasia the paralysis of the speech function is the result of organic disease, in others of functional or nutritional changes. Any lesion which destroys or interrupts the function of the speech centres in the cerebral cortex, of the conducting tracts which pass to or from these speech centres, or of the connecting fibres or tracts which pass between these speech centres, may produce aphasia. The organic lesions which are the most common causes of aphasia are—embolic or thrombotic plugging of the nutrient arteries, hæmorrhagic extravasations, cerebral tumours, and cerebral abscesses. In the case of embolic plugging and hæmorrhagic extravasation the aphasic symptoms are suddenly produced; in the case of thrombotic plugging the aphasic symptoms are usually produced quickly (in the course of a few hours), but not so suddenly and rapidly as in embolism and hæmorrhage; in the case of abscess and tumour the aphasic symptoms are usually produced still more gradually.

The more common causes of temporary or functional aphasia are—temporary anæmia of the speech area, reflex inhibition of the speech centres, the cortical exhaustion following an epileptic fit, a congestive attack in general paralysis of the insane, or an attack of migraine; and the temporary changes, whether of a functional or structural kind, which are developed in the cerebral tissues in the course of hysteria, chorea, uræmia, lead poisoning, alcoholic poisoning, general debility, etc. The usual form of temporary (functional) aphasia is inability to speak (temporary motor-vocal aphasia); but temporary word-blindness with hemianopsia is probably not uncommon. Temporary word-deafness is probably much more rare. Temporary paraphasia and temporary agraphia or paragraphia occasionally occur.

THE CLINICAL FEATURES OF APHASIA

THE SYMPTOMATOLOGY OF WORD-DEAFNESS (AUDITORY APHASIA).—In typical and uncomplicated cases of complete word-deafness (inability to understand spoken language) the patient is not deaf (though in some cases there is some impairment of hearing); he can hear ordinary sounds and noises, such as the ticking of a watch; but he is unable to intelligently comprehend speech sounds (words, spoken language). He is in the position of a man who is spoken to in a language with which he is absolutely unacquainted; he hears the words as sounds, but he has no knowledge of their meaning as speech sounds.

Word-deafness in a complete form is, in my experience, the rarest form of aphasia. In many cases the word-deafness is incomplete, the degree of the defect being very variable in different cases.

The exact character of the symptoms varies with the position of the lesion—whether it is subcortical or cortical.

The symptoms characteristic of subcortical word-deafness.—When the lesion merely involved the ingoing (afferent) fibres which pass to the auditory speech centre, the only results are—(*a*) word-deafness (inability to understand spoken language); (*b*) inability to repeat spoken language; and (*c*) inability to write from dictation. In cases of subcortical auditory aphasia the "*way-in for spoken speech*" is blocked; the auditory speech centre is cut off from the external world, but its internal activity remains, and it is still in (direct functional) connection with the other speech centres (motor and visual), and with the other centres throughout the cerebral cortex, which, inclusive with it, we may, for the sake of convenience, collectively term the seat of consciousness, intelligence, and the will. Consequently in cases of subcortical auditory aphasia there is no obliteration of the memories of auditory speech; thought and internal speech are not interfered with; and there is no disturbance produced in the action of the associated speech centres, there is no word-blindness, and the patient has no difficulty in spontaneously expressing himself either in spoken or written speech or in reading aloud. The ability to spell is not impaired. Cases of *subcortical* (*pure, isolated,* or *subpictorial*) word-deafness are extremely rare.

The symptoms characteristic of cortical word-deafness.—When the lesion involves the auditory speech centre itself the speech alterations are much more profound. The memories or images of auditory speech (spoken words) are more or less completely obliterated; internal speech and thought are seriously interfered with; the ability to spell is markedly impaired; and modifications more or less marked and profound are usually (? always) produced in the action of the motor-vocal and visual speech centres, and in the action of the motor-writing centre. In typical and complete cases of cortical word-deafness the patient is not only word-deaf, unable to repeat spoken language, and unable to write from dictation, but there are usually some defects in his power of expressing himself spontaneously in spoken speech (for the function of the motor speech centre is largely stimulated by and controlled by the action of the auditory speech centre); in his ability to understand written speech (for the action of the visual speech centre is intimately associated with, and to some extent dependent upon, the action of the auditory speech centre); and in his ability to write spontaneously. He usually makes some mistakes in voluntary (spontaneous) speech, in reading, and in writing, and in some cases the mistakes are very marked. The commonest form of mistake is to apply wrong names to objects without being conscious of the mistake, and to make use of roundabout expressions (*paraphasia*). In some cases, though the patient makes use of a copious vocabulary, his speech is altogether unintelligible (*jargon*). In rare cases, and this probably only occurs as a temporary condition, immediately after the onset of the attack, the patient may not be able to speak at all. In some cases of word-deafness in which the word-deafness is incomplete, the patient (who is unable to understand a spoken word or sentence) is conscious of his mistakes, possibly in consequence of the additional information received in the act of articulation and vocalisation. In the great majority of cases of marked word-deafness the patient is unable to repeat what is said to him. In rare cases he can repeat like a parrot (echo speech) what is said to him, although he cannot understand spoken speech. In some cases of word-deafness the power of appreciating music is retained, in others it is lost.

In cases of word-deafness there is not usually any paralysis of the limbs or face, though a certain degree of temporary hemiplegia is not uncommon at the onset of the attack. Hemianopsia is not present, unless of course the lesion is so extensive as to involve the optic radiations of Gratiolet, or unless there should happen to be a second and independent lesion causing the hemianopsia.

THE CHIEF CHARACTERISTICS OF MOTOR APHASIA FOR SPOKEN SPEECH (MOTOR-VOCAL APHASIA, or APHEMIA).—In typical and marked cases of motor-vocal aphasia the patient is unable to express himself by vocal speech. He can neither speak spontaneously, repeat vocal speech, nor read aloud; but he is not absolutely wordless (dumb) unless *both* motor-vocal speech centres are entirely destroyed. He can usually utter some words, such as *yes* and *no*, intelligently, and other words in a reflex, automatic, or emotional way; but he cannot intelligently and of set purpose innervate the muscular organs concerned in the production of articulate language in the way which is necessary for the production of intellectual, as distinct from emotional or merely automatic language. As Hughlings Jackson has expressed it, the patient is *speechless* but not (usually) *wordless*.

The symptoms characteristic of subcortical motor-vocal aphasia.—In cases of subcortical (*pure, isolated*, or *subpictorial*) motor-vocal aphasia the patient is able to understand what is said to him (the auditory speech centre being unaffected); he is able to read printed and written characters (the visual speech centre being unaffected); he can, provided that the fibres passing from the motor-writing centre are not at the same time implicated, express himself in writing; but he cannot express himself by means of spoken language. He can neither speak spontaneously, repeat what he is told to repeat, nor read aloud. In cases of *subcortical* motor-vocal aphasia, it is merely *the "way out" for vocal speech* which is blocked; internal speech is entirely unaffected. The patient knows what he wants to say, but he cannot say it. He can, provided that his intelligence is unaffected, indicate by signs the number of syllables in the words which he wishes to express, but he is unable to produce speech sounds (to speak).

The symptoms characteristic of cortical (*true, or pictorial*) *motor-vocal aphasia or aphemia.*—In cases of cortical motor-vocal aphasia, in which the lesion involves the motor-vocal speech centre itself (Broca's convolution) the patient is unable to speak spontaneously, to repeat what he is told to repeat, or to read aloud; but, in addition, the power of understanding spoken speech is sometimes (always, according to Déjerine) impaired, though usually only in a very slight degree; the power of understanding complicated written passages is sometimes (? always) impaired; and there is usually, though not necessarily, agraphia, the defect in writing being usually proportionate to the defect in speaking. Even when the intelligence is sufficiently intact to enable the patient to appreciate the nature of the test, he is usually (though I do not think invariably) unable to correctly indicate the number of syllables in a word (Proust-Lichtheim test). Cases of cortical motor-vocal aphasia differ very notably in the degree of their severity.

In many cases of aphasia in which the patient is absolutely unable to make use of *intellectual* language, he retains the use of those emotional expressions ('Oh!" "Oh dear!" "Oh my!" "By Jove!" etc.), which in his particular case have become mere explosive or reflex utterances, devoid of any real meaning or intellectual value. It is important to remember that the monosyllables *yes* and *no* may be used either reflexly or intellectually; as Ross well puts it, "The unit of emotional language is, so far as it is expressed in vocalisation, an exclamation or an interjectional word or phrase, but the unit of intellectual language is a proposition, and if the monosyllabic reply of an aphemic patient possess any speech value, it must be equivalent to a distinct proposition." It is supposed by some authorities that in those cases of aphasia in which emotional expressions are retained, they are stored up, as it were, in, and discharged from, the inactive motor-vocal speech centre on the unaffected ("non-driving") side of the brain, or in some lower centre, say in the pons Varolii or medulla oblongata. In some cases of motor-vocal aphasia the patient retains the power of using a certain expression or sentence only. In some cases the *recurring utterance* is an expression which has become habitual to him, *i.e.* is automatic; in other cases, the expression or sentence which is retained is one which apparently was last used, or was just about to be used, when the patient was attacked with the aphasia. "It is not improbable that words spoken, or about to be spoken, during a period of great excitement, will leave permanent traces on the organisation of the brain, which renders them liable to be subsequently uttered as interjectional phrases during emotional states" (Ross). Further, in some cases of motor-vocal aphasia the patient can, in singing, more or less clearly articulate and pronounce words which he is unable to speak.

In cases of motor-vocal aphasia, whether cortical or subcortical, right-sided hemiplegia is usually present. In rare cases, in which the arterial branch which supplies Broca's convolution is alone plugged, there is no motor paralysis, except perhaps a temporary paralysis (due to temporary shock and disturbance of the circulation) in the face and arm, and it may even be in the leg on the opposite side.

THE CHIEF CHARACTERISTICS OF WORD-BLINDNESS (ALEXIA OR VISUAL APHASIA).—In typical and marked cases of word-blindness (inability to understand written and printed language) the patient is able to see quite well, in other words, central vision is usually unimpaired, though right-sided bilateral homonymous hemianopsia is often present; but he is unable to understand written speech characters. He is in the position of a person who is asked to read an unknown tongue, written in symbols (letters and words) which he has never seen before. In word-blindness, as in other forms of aphasia, the severity of the aphasic defect varies greatly in different cases. In some cases the patient is unable to recognise and name the individual letters; in other cases he can name some, or it may be almost all of the individual letters, but he is unable to read combinations of letters (words and sentences); in others he can read some words (combinations of letters), but he is unable to read (name) the individual letters of which they are composed. In some cases the patient can read figures; in others he cannot. In some cases the patient is unable to name objects; but this, which is undoubtedly in some cases a form of *visual* aphasia, is not, strictly speaking, a form of *word*-blindness. In some cases the ability to read music is retained; in others it is lost. In some cases the functions of the other speech centres (auditory and motor-vocal speech centres) are impaired, though probably rarely in a marked degree; in others, unaffected.

The clinical characteristics of subcortical (pure, isolated, or subpictorial) word-blindness.—In *subcortical* word-blindness *the "way-in" for visual speech impressions* is blocked; consequently the patient is unable to understand written speech or to read aloud. [The "block" is situated between the half vision centres and the visual speech centre, and it is important to note that subcortical word-blindness is *only* produced when the fibres passing from *both* half vision centres are implicated. Complete destruction of the fibres passing from *one* half vision centre to the visual speech centre does not produce word-blindness; under such circumstances the patient receives the (visual) information, which is necessary to enable him to read, through the half vision centre and the fibres which are sound (unaffected)]. In cases of subcortical word-blindness the patient can, however, write spontaneously and from dictation; but he cannot, after an interval has elapsed, read what he has written; though in some cases he is able to decipher the meaning of written speech (which he cannot read) by tracing the letters over with his finger or a pencil; in other words, he is able, in some cases, to ascertain the meaning of written speech characters by the aid of the kinæsthetic impressions which attend the muscular movements involved in copying the written characters (letters and words) which he cannot read in the ordinary way (by vision). In cases of subcortical word-blindness internal speech and thought are not impaired (since the action of the auditory and motor-vocal speech centres is not deranged). Though the patient is unable to read (name) the individual letters by the sense of sight, he retains the mental conception of the form and shape of the letters, and he may be able to describe their form in words; he is able to write from dictation, and he may be able to write on a typewriter, but he is unable to *intelligently copy*, *i.e.* to transcribe printed into written characters, though he is usually able to *mechanically* copy printed or written speech characters, *i.e.* stroke for stroke, as he would copy a map or a drawing.

The lesion being situated far back (*i.e.* in the white matter below the angular gyrus or in the anterior end of the occipital lobe) there is not usually any motor paralysis, though right-sided hemiplegia may be temporarily present immediately after the onset. There may, however, be some hemianæsthesia on the opposite (right) side. Hemianopsia and hemiachromatopsia are usually (always) present.

The clinical characteristics of cortical (true or pictorial) word-blindness.

—*In cortical* word-blindness the patient is not only unable to understand written language and to read aloud, but he is also unable to write spontaneously and (with rare exceptions) to write from dictation. He can *mechanically* copy written or printed speech characters, *i.e.* as he would copy a drawing or a map (*literally*, stroke for stroke, line for line); but he cannot *intelligently* copy written speech characters; he is unable to transpose typewritten characters into manuscript, and *vice versâ*. He cannot arrange separate printed (block) letters in the form of words, and he cannot write on the typewriter. He has lost the mental conception of the form and shape of the letters, and of the manner in which the individual letters must be arranged and grouped for the formation of written words.

In cases of cortical word-blindness there is usually (? always) some derangement, latent or obvious, in the understanding of spoken language, and often some derangement of spoken speech, usually paraphasia. The ability to spell is usually impaired, at all events in some degree.

Right hemiplegia is usually absent, and right-sided hemianæsthesia is less frequently present than in the subcortical variety.

THE CHIEF CHARACTERS OF AGRAPHIA (MOTOR-WRITING APHASIA, OR GRAPHIC APHASIA, as it is sometimes termed).—There is much difference of opinion as to the existence of a separate writing speech centre, and as to the nature of agraphia. Two views are held, viz.—(1) That a separate and distinct writing *speech* centre exists; according to this view agraphia is *aphasia of the hand*. (2) That a separate writing speech centre does not exist, and is in fact unnecessary; according to this view the writing centre is merely a part of the ordinary psycho-motor centre for the upper extremity (hand, wrist, etc.) The space at my disposal in this article does not permit me to discuss the question. The balance of evidence seems to me to be in favour of the latter view.

In some cases agraphia is associated with sensory aphasia, more especially with word-blindness, and, in others, with motor-vocal aphasia. Up to the present time no well-authenticated case has been recorded in which isolated agraphia—agraphia, and nothing but agraphia—has resulted from a localised lesion in any part of the motor area of the cortex.

In most cases in which agraphia is associated with cortical motor-vocal aphasia the agraphia is, roughly speaking, proportionate to the vocal speech defect; but this is not invariably the case.

In those cases in which the agraphia is complete and unassociated with motor-vocal aphasia word-blindness is almost always, but not necessarily always, present; for a lesion which destroys the conducting fibres which pass from the visual speech centre to the motor-writing centre, but which does not involve either of these centres, may, I believe, produce agraphia.

In those cases in which the *agraphia is associated with cortical word-blindness* the agraphia is usually (? always) complete. The patient can neither write spontaneously nor from dictation, he is unable to copy intelligently (to transcribe type into manuscript), to write on the typewriter, and, in most cases, to write with his left hand.[1]

In those cases in which the *agraphia is associated with subcortical word-blindness* the agraphia is incomplete. Although the patient cannot intelligently copy or transcribe type into manuscript, he is able to write spontaneously (though after a short interval he is unable to read what he has just written), and he may be able to write from dictation.

Complete destruction of the auditory speech centre, or rather, it would be more correct to say, of both auditory speech centres, ought theoretically to produce inability to write spontaneously and from dictation (since there seems good reason to believe that in spontaneous writing the auditory speech centre is first stimulated—in other words, since the idea of the word appears first to take concrete shape in the auditory speech centre). But it is probable that in some persons who are accustomed to read and write much, *complete* destruction of the auditory speech centre does not produce complete agraphia, though it does (usually) produce paragraphia.

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[1] "Mirror-writing" will be treated under its own heading.

TACTILE APHASIA.—There is every reason to suppose that blind people, who have learned to read and write by the sense of touch, are occasionally affected with what may be termed *tactile* aphasia; but, so far as I know, no case of the kind has been actually recorded.

COMBINED FORMS OF APHASIA.—Cases of isolated aphasia (uncomplicated word-deafness, uncomplicated motor-vocal aphasia, uncomplicated word-blindness, and uncomplicated agraphia) are probably less frequent than cases of aphasia in which more than one of the speech faculties is involved and deranged. This is due to the following causes: (1) That the subcortical sensory aphasias are more difficult to produce than the cortical forms; for, to produce subcortical word-deafness or subcortical word-blindness, the lesion must interrupt *all* of the afferent fibres passing to the auditory speech centre or the visual speech centre from the lower auditory or visual centres respectively; whereas the cortical forms of word-deafness and word-blindness may be produced by localised lesions which destroy the auditory or visual speech centres respectively. (2) That the actions of the different speech centres are, to a large extent, mutually dependent one upon another; consequently a lesion in one of the cortical speech centres will (usually) produce more or less marked derangement of the action of the others. These derangements are, I think, usually most marked when the auditory speech centre is involved; much less marked (except so far as writing is concerned) when the visual speech centre is involved; very slight, and in many cases not obvious, when the motor-vocal speech centre is involved; and not produced at all when the motor-writing centre is involved (though I speak with hesitation regarding the function of the visual speech centre). (3) That the two sensory speech centres (the auditory speech centre and the visual speech centre) on the one hand, and the motor-vocal speech centre and the motor-writing centre on the other, are situated in close juxtaposition and continuity. Hence a lesion which involves the auditory speech centre is very likely at the same time to involve the visual speech centre, and *vice versa*; and a lesion which involves the motor-vocal speech centre is very likely at the same time to involve the motor-writing centre, and *vice versa*. (4) That all of these speech centres are supplied by the same artery (the middle cerebral), and that the branches which supply the auditory speech centre and the visual speech centre on the one hand, and the branches which supply the motor-vocal speech centre and the motor-writing centre on the other, come off in close juxtaposition. Hence the frequency with which word-deafness and word-blindness on the one hand, and motor-vocal aphasia and agraphia on the other, are combined in those cases in which the aphasia is due (as it most frequently is) to softening, the result of arterial obstruction, whether embolic or thrombotic.

TOTAL APHASIA.—The fact that all of the great speech centres are supplied by the same (middle cerebral) artery also, of course, explains the occurrence of *total* aphasia—a combination of word-deafness, word-blindness, motor-vocal aphasia and agraphia—when the main trunk of the middle cerebral or Sylvian artery is plugged by an embolon or is thrombosed.

COMMISSURAL APHASIA.—To provide for the intimate associations which exist between the different speech centres, connecting and commissural fibres are, of course, necessary; and it cannot be doubted that a variety of aphasic symptoms may be due to interruption or destruction of these connecting fibres, the speech centres themselves, the afferent fibres proceeding to the sensory speech centres, and the efferent fibres passing off from the motor-vocal speech centre and the motor-writing centre being unaffected by the lesion. These *connection, commissural,* or *conduction* forms of aphasia, as they are sometimes termed, probably differ in some particulars from the cortical and subcortical forms of aphasia which have already been described; but I doubt whether, in the present state of our knowledge, it is advisable to regard them as *distinct forms* of aphasia; their characteristic features (if they have characteristic features) are as yet unknown.

THE ASSOCIATION OF APHASIA AND MOTOR PARALYSIS.—The frequent association of aphasia with motor paralysis (aphasia and right hemiplegia in right-handed persons, and of aphasia and left hemiplegia in left-handed persons) is, of course, due to the fact that the middle cerebral artery supplies not only the speech centres, but the motor area of the cortex.

DIAGNOSIS

Aphasia is a symptom, or rather a group of symptoms, not a disease; consequently in the diagnosis of a supposed case of aphasia we must endeavour to determine, in the *first* place, whether the speech defects which are present are actually aphasic in character; in the *second* place, what is the position of the lesion (anatomical diagnosis); and in the *third* place, what is the pathological character of the lesion (pathological diagnosis).

In the definition of aphasia which has been given at the beginning of this article I have stated that, in my opinion, only those speech derangements should be included under the term aphasia which are due to functional disturbance or organic disease of the higher cerebral speech mechanisms properly so-called. In my opinion the term aphasia should not include defects in the perception of spoken and written speech due to disease in the end-organs of special sense (the ear and the eye); it should not include mere defects of intelligence, defects of pantomime, nor mere defects of articulation. Provided that these points are kept in view, there is rarely any difficulty in deciding whether the speech defects in any particular case of nervous disease are actually aphasic or not.

ANATOMICAL DIAGNOSIS.—The position of the lesion which is the cause of the aphasia can usually be determined with considerable accuracy. Except in rare instances, there is no difficulty in deciding whether the lesion is situated in the left or the right hemisphere of the brain by ascertaining and observing: (1) whether the patient is right-handed or left-handed (this is of first importance); and (2) whether the associated cerebral symptoms of a non-aphasic kind (motor paralysis, hemianæsthesia, hemianopsia, etc.) are right-sided or left-sided.

Having determined that the lesion is in the left or the right hemisphere of the brain, we have next to determine whether the chief (the primary or initial) speech disturbance is indicative of a lesion of the auditory speech centre, the motor-vocal speech centre, or the visual speech centre. A correct conclusion on this point can generally (though not invariably) be arrived at by a careful analysis of the speech defects which are present.

The further diagnostic question, whether the lesion is cortical or subcortical, can also in many cases be determined with considerable accuracy. (See the points of difference between the cortical and subcortical varieties of word-deafness, motor-vocal aphasia, and word-blindness respectively, which have already been detailed.

PATHOLOGICAL DIAGNOSIS.—The following questions have, if possible, to be determined:—(1) Whether the aphasia is the result of functional derangement or organic disease; (2) if due to functional derangement, what is the exact nature of the functional disturbance; and (3) if due to organic disease, what is the exact nature of the organic lesion. These questions involve the differential diagnosis of the various functional conditions and organic lesions which may cause aphasia (cerebral embolism, cerebral thrombosis, cerebral hæmorrhage, hysteria, migraine, etc.) For a discussion of the subject the reader is referred to other parts of this work.

PROGNOSIS

Since the conditions which are included under the term aphasia are merely symptoms, the prognosis as regards the risk of death, the risk of recurrence of the lesion, etc., has to be based upon the nature of the lesion which is the cause of the aphasia, the severity of that lesion in the particular case, and the numerous factors which go to form the personal equation. (See the functional derangements and organic lesions which may produce aphasia in other sections of this work.)

The prognosis as regards the aphasic symptoms *per se* has to be guided, in the *first* place, by the consideration whether the aphasia is due merely to a functional derangement, or whether it is due to an organic lesion (this is a question of diagnosis); and if the aphasia is the result of organic disease (*a*) the nature of the lesion; (*b*) the severity of the lesion; and (*c*) the capabilities or probabilities of *compensation*. In trying to determine these questions a variety of considerations, amongst which the following are of most importance, have to be taken into account, viz. the age and general vitality of the patient; the condition of his cerebral tissues, and especially of his cerebral arteries; the nature of the lesion which is the cause of the aphasia (whether embolism, syphilitic thrombosis, atheromatous thrombosis, cerebral hæmorrhage, cerebral tumour, cerebral abscess, etc.); the form of the aphasia (word-deafness, for example, is probably in most cases more easily recovered from than word-blindness); the severity of the aphasia; the length of time which the aphasic symptoms have lasted, and the degree of improvement which has already taken place; the cortical or subcortical nature of the lesion (as a rule, the prognosis, as regards the aphasia itself, is more favourable in the subcortical than in the cortical forms); the effects of treatment; the presence or absence of associated lesions (in the heart, kidneys, etc.)

TREATMENT

In cases of aphasia two objects have to be kept in view, viz.:—(1) *The treatment of the functional derangement or organic lesion on which the aphasic condition depends.* This, which is by far the most important object, has to be guided by the nature of the cause of the aphasia (see the treatment of the various functional conditions and organic lesions which may cause aphasia, as set forth in other parts of this work); and (2) *The treatment of the aphasic condition itself.* Since the different conditions which are grouped together under the common term aphasia may be regarded as paralyses of the sensory or motor speech mechanisms, the same principles which guide the treatment in ordinary cases of motor and sensory paralyses should be carried out. The cause of the aphasia (paralysis) must if possible be removed, the general health must be brought into the best possible state of efficiency, and every effort must be made to restore the function of the damaged parts, and to promote compensation and the restoration of the speech function by training and developing the function of the corresponding speech centres on the non-active ("non-driving") side of the brain. In cases of complete and total aphasia the patient is, as we have seen, reduced, so far as his speech functions are concerned, to the position of a young child who is unable to understand spoken language, to speak, to read, and to write. In the treatment of such a case the same methods of education and training must be employed which are employed in the training and development of the speech faculties in the young child. And in cases of isolated or partial aphasia (word-deafness, motor-vocal aphasia, word-blindness or agraphia) the same principle must be carried into effect, viz. the training, after Nature's method, of the particular speech faculty which is damaged or impaired. (See Physiological Introduction, p. 294.) In cases of motor-vocal aphasia, as in cases of ordinary motor paralysis, the patient should be encouraged to practise, regularly and frequently, speech movements. In cases of word-blindness and agraphia the patient must for a second time be taught to read and to write.

THE MEDICAL JURISPRUDENCE OF APHASIA

APHASIA AND WILL-MAKING.—In cases of aphasia important medico-legal questions, such as the ability of the patient to manage his affairs, to make a will, etc., every now and again arise. The following is an outline of the more important points which arise in connection with these questions:—

Speaking generally, it seems to me that from a medical point of view, if an aphasic patient is able to make a will, he is, in most cases, at all events, so far as his mental condition alone is concerned, qualified to manage his affairs; though in many cases, owing to the aphasic condition (the difficulty in communicating with the patient which the aphasic condition entails, and the paralytic condition with which the aphasia is so often associated), it will be convenient and advisable, more especially when his affairs are complicated, that they should be managed for him.

It is impossible to make any general statement regarding the will-making power of aphasic patients which can be applied to all cases. Some aphasic patients are able to manage their affairs and to make a will, others are not; *each case must be decided on its own merits.* There is, as Dr. Hughlings Jackson has pointed out, no single well-defined entity—loss of speech or aphasia. Hence the question, "Can an aphasic make a will?" cannot be answered any more than the question, "Will a bit of string reach across this room?" can be answered. The question should be, "Can this or that aphasic patient make a will?"

In connection with the will-making power of aphasic patients the following points must be kept in view:—

(1) That several different varieties of speech defect are included under the term aphasia, namely, word-deafness, word-blindness, vocal-motor aphasia, and agraphia; and that the will-making capacity is by no means the same in these different forms or varieties. Hence in each particular case it is necessary to determine the exact form of aphasia which is present.

(2) That it is necessary to draw a distinction between the subcortical aphasias on the one hand, and the cortical aphasias on the other; for, in the subcortical forms, thought and internal speech are usually unaffected,

whereas, in the cortical forms, internal speech and thought are usually in some degree, and often profoundly, affected.

(3) That in some cases of aphasia these different varieties of speech defect are met with in an isolated form, whereas in other cases they are combined.

(4) That different cases of aphasia differ, not only as regards the nature of the speech defect, but also in degree—that is, as regards the severity of the special speech disturbance by which they are characterised.

(5) That in some cases of aphasia there is great difficulty in communicating with the patient (in making him understand the questions which we desire to put to him); and that in other cases the patient has great difficulty in communicating with us (in making us understand his wishes and ideas); and that in some cases both of these defects are combined.

(6) That in some cases of aphasia the intellectual faculties are little, if at all, impaired, while in others they are profoundly altered.

(7) That in many cases of aphasia the emotional faculties are markedly disturbed; and that in some cases, as the result of the brain disturbance—the brain lesion on which the aphasia depends, or with which it is associated—the judgment and affections of the patient are perverted, so that he takes unreasonable likes and dislikes to relatives, friends, persons who are in contact with him, etc.

It will be readily understood that in consequence of these circumstances there is, in many cases of aphasia, very great difficulty in forming a correct estimate of the mental condition of the patient and of his will-making power.

Further, the capacity of patients affected with aphasia for making a will largely depends upon the nature of the will, whether simple or complicated; and is especially difficult when the patient has not, prior to the development of the cerebral lesion, thought out the manner in which he wishes to dispose of his affairs (the details of his will), or given any instructions to his lawyer regarding his will.

It is hardly necessary to say that it is essential in the interests of justice and of the patient's representatives and heirs that the most scrupulous care be taken not to suggest to the patient what he should do (how he should dispose of his property, to whom he should dispose of it, etc.). No attempt should be made to obtain the patient's instructions for a draft will or to get him to execute a will immediately after the occurrence of an aphasic attack, or just after a relapse; for under such circumstances the brain action is necessarily disturbed in a greater or less degree. The medical attendant should be present when an aphasic makes a will, with the object of assisting, by his skilled knowledge and advice, the lawyer to ascertain the wishes and desires of the patient, and in order to satisfy himself so that he may be able to testify that the patient was, *at the time when the will was made*, mentally capable of making a will, that the instructions for the will were correctly elicited, and that the will was correctly executed. The lawyer should consult the medical attendant as to the mode of procedure (the best method of ascertaining the wishes and desires of the patient), the length of each examination, etc.

I may further add that great patience, in some cases repeated examinations (for, as has been stated, the brain power of aphasic patients is easily exhausted), and the most scrupulous fairness and exactness in eliciting the facts and in drawing conclusions as to the significance of the facts, are absolutely essential in order to form a true and correct estimate of the patient's mental condition and of his will-making capacity. Further, it must be remembered that there are many sources of fallacy. For example the recurring, emotional, or automatic utterances of patients affected with aphasia may easily be taken for intellectual expressions (propositions), whereas they may have no intellectual value whatever. Again, if the patient is able to answer simple questions by means of signs (pantomime), the physician may, not unnaturally perhaps, but far too hastily, conclude that the reasoning power and intellectual faculties are altogether unaffected.

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Aphonia. *See* VOICE.

Aphrodisiacs.—These are remedies which increase sexual desire or power. The term may be used to include not only drugs and other measures which act upon the sexual organs, upon neighbouring structures, especially the urinary tract, and upon the nervous system, but also tonic treatment of every kind, whether medicinal, hygienic, or dietetic.

ENUMERATION.—The principal aphrodisiacs are—alcohol, camphor (at first), cannabis indica, castor and musk, damiana, nux vomica and strychnine, opium, phosphorus; general tonics; a rich, especially a nitrogenous diet, and bodily and mental rest.

MODE OF ACTION AND USES.—Aphrodisiacs are not drugs to be used symptomatically or indiscriminately. To stimulate sexual desire, for example, in a patient suffering from the effect of excessive venery, would prove a dangerous experiment. Every case of weakened sexual desire or power must be carefully examined, to determine what causal conditions are at work, and treated accordingly. In all cases, the possibility of the symptom having some relationship to organic disease must be borne in mind.

The part any aphrodisiac remedies may play in the treatment of a particular case must be gathered from a consideration of their various modes of action. Fresh air, an abundant and varied diet with plenty of fresh meat and some tonic, abstinence from severe muscular exertion, and from mental work and worry, all play an important part in treatment. Nux vomica and strychnine are perhaps the drugs most frequently used, not only on account of their general tonic properties, but on account of their special action on the spinal cord.

Many substances which irritate the genito-urinary tract, especially substances belonging to the oleo-resin group, have an aphrodisiac effect; and cantharides, used cautiously, act in the same manner.

Cannabis indica is largely used in the East. It stimulates the cortex and gives rise to dreams of a sexual character. It is apparently often combined with other drugs, such as cantharides, which produce a local effect on the urinary tract, and so reflexly stimulate the genital organs.

Alcohol powerfully stimulates sexual desire, but at the same time weakens the sexual act, so that coitus is apt to be imperfect.

Opium is not used as an aphrodisiac, but it is important to remember its effect in this way, in treating the subjects of the morphia habit. Sexual desire in such patients, especially when they have been for some time under treatment, may be very strong, and as the power of self-control is greatly diminished, it is very important that male patients should be attended by male nurses.

Aphthæ. *See* STOMATITIS.

Apoplexy. *See* CEREBRUM.

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APPENDICITIS

THE term *appendicitis* is one which, originating in America, is now applied to all those affections which are comprised under the names of *typhlitis, peri-typhlitis, epi-typhlitis, para-typhlitis*, etc. All have their origin in the appendix, and it is very questionable whether a case of inflammation about the cæcum, not due to tuberculous or cancerous disease, ever occurs without being secondary to disease of the appendix.

Typhlitis, as a disease *per se*, that is originating in the cæcum, does not occur except very rarely; I myself have never seen such a case. Some thought, until quite recently, that stercoral cæcitis was exceedingly common, and now most people doubt whether it ever existed, and it is unknown to pathologists. In my long experience I have never yet seen at a post-mortem, in the dissecting-room, or at an abdominal operation, such a thing as impaction of fæces in the cæcum, or even a loaded cæcum. Occasionally small scybalous masses may be felt moving freely about, and liquid fæces may be occasionally met with, but unless there is some obstruction of the bowel lower down, from malignant or other disease, the cæcum is nearly always empty. Impaction of fæces in the cæcum has been recognised by clinicians, never by pathologists or abdominal surgeons. Again, as Hawkins remarks, if fæcal impaction ever takes place in the cæcum, why should this set up an inflammatory condition more than fæcal impaction in the sigmoid flexure—a much more common site for such an occurrence?

Peri-typhlitis, para-typhlitis, and *epi-typhlitis* are terms which are now almost obsolete, and are quite unnecessary; they refer to the situation, not the origin of the inflammation, for they all owe their origin to a primary inflammation of the appendix. The position of the appendix will determine the site of the surrounding inflammation, whether beneath, above, or alongside the cæcum. It was formerly thought that perityphlitis was due to an inflammation of the cellular tissue behind the cæcum. Now that we know that the cæcum almost invariably floats free, and is covered by peritoneum on all sides, behind as well as in front, inflammation of the cellular tissue around it is an impossibility.

In all inflammations about the cæcal region inspect the appendix, the fault will almost invariably lie with it. The appendix may be seriously diseased, even should it appear externally perfectly healthy.

Anatomy.—The appendix is a degenerated ancestral cæcum, and is situated at the posterior and lower part of the cæcum. In early fœtal life it is in direct continuation with the cæcum, and seems to be its tapering extremity; this form of cæcum and appendix, in a small percentage of cases, persists, and is situated higher up than usual, but generally the appendix has an inner and posterior position, because in development the anterior part of the cæcum grows faster than the posterior, and hence causes the part which was lowest to be posterior. The opening of the appendix into the cæcum is near the ileo-cæcal valve. The three longitudinal bands which run down on the cæcum meet at the appendix, and are continued down upon it.

The appendix measures about three and a half inches in length; it may only measure half an inch, and again may be six inches in length, and it has been reported absent. The writer on one occasion removed a process which measured nine inches.

The average width of the vermiform process is a quarter of an inch; it has connected with it a mesentery (meso-appendix) triangular in shape, and this contains blood-vessels and nerves—the artery runs along the free edge of the mesentery, which is shorter than the border in contact with the process, and from this cause the appendix is curled up on itself; in many adults the mesentery does not reach to the end of the process, which is quite free and covered by peritoneum.

The usual position of the appendix is upwards and inwards, pointing toward the spleen and behind the cæcum and its mesentery. In many cases it hangs over the brim of the pelvis, in others again it is situated posterior to the cæcum, having a direction towards the right kidney. It may be hidden away in one of the ileo-cæcal fossæ.

The positions in which the appendix may lie are very various. In cases of floating cæcum, where the ascending colon has a long mesentery, it may be anywhere—in the left iliac fossa, pelvis, near the umbilicus, and on one occasion, during an operation for appendicitis, the writer saw it above the stomach and attached to the lesser omentum.

The lumen of the appendix varies considerably, being much greater in young people than in old—Ribbert asserts that the cavity of the vermiform process tends to undergo obliteration; 400 cases were examined, and in 25 per cent the process was more or less obliterated. In 50 per cent of the obliterated cases its distal fourth only, and 3·5 per cent the whole process was a solid cord. This tendency to obliteration increases with age, and may be regarded as a degenerative process.

STRUCTURE.—Like the intestine it has an external, serous, and peritoneal coat, two muscular coats, outer longitudinal, inner circular, a submucous and a mucous layer. In the mucosa are a number of lymphoid follicles and tissue resembling somewhat that found in the tonsil. These lymphoid glands vary much in number, and where there is a tendency to obliteration of the cavity the lymph follicles are few in number or absent altogether. The extreme end of the appendix is usually made up of fibrous tissue, and is often bulbous—the lymphatics are arranged as in the intestines, and are connected with lymphatics of the small intestine. Its single artery, which runs along the free border of its mesentery, is derived from the ileo-colic, and distributes its branches to the process as it goes.

Besides its proper mesentery there is a fold from the ileum to the meso-appendix which is devoid of blood-vessels, and is the remains of the original mesentery (Treves). It is held by some that any displacement or distension of the cæcum would make tense this fold and tend to cause a kinking or twisting of the appendix and thus produce congestion, tumefaction, and catarrhal inflammation.

ÆTIOLOGY.—Being the organ which is a rudiment, and, so to speak, functionless, its vitality is low and its resisting powers not sufficient to cope with its very unfavourable surroundings. Its dependent condition and the fact that it communicates with the large bowel by a narrow orifice, and this protected often by a valve which prevents its contents getting free exit, predisposes in every way to inflammatory attacks, especially as micro-organisms are always present.

Appendicitis occurs at all ages, but most commonly between twenty and thirty; it may occur in the infant of a few months old and in old people over eighty. It is more common in males than in females. Dr. Geo. Armstrong has collected 517 cases, in three the sex was not mentioned, in the remaining 514, 340 were males and 174 females; 209 cases were between the age of twenty and thirty; other observers have found this affection more common between ten and twenty years. The probable reason that males are more liable to this disease than females, is that males are more subject to traumatism, for persons who lift heavy weights and undergo much muscular exertion are more subject to the disease. Heredity also has some influence, for it is not uncommon to see two, three, or even four cases in one family.

Errors and indiscretions of diet are apt to induce an attack, and one frequently is told by patients that the attack came on after eating a hearty meal or something that disagreed with them. This is especially the case in the form known as "recurrent appendicitis."

Osler states that he has seen a number of cases in boys who have gorged themselves with peanuts, a favourite diet with the juveniles in the United States. It is held by some that those who have a rheumatic diathesis are more liable to the disease, but such has not been my experience. The appendicular lymphoid tissue has been compared to that of the tonsil.

Typhoid and tuberculous ulcerations of the appendix sometimes occur, and it is impossible to tell them from the ordinary inflammatory cases. Malignant disease may also involve this part of the bowel as well as any other, and may often extend from ileo-cæcal cancer.

The presence of fæces in the appendix is not uncommon, and these may fill the lumen so that on squeezing the process a mould of fæces exudes. Enteroliths and fæcal concretions are very frequent; how these are formed is still uncertain, micro-organisms are supposed to have something to do with their occurrence; they may look like date or cherry stones, beans, seeds, and from this resemblance the supposition came that foreign bodies were frequently entrapped in the appendix. If these concretions be allowed to dry, they can be easily crushed between the thumb and forefinger. These cause erosions and ulcerations of the mucous membranes, and so allow micro-organisms to infect the deeper tissues and produce an acute inflammatory condition leading ultimately to peritoneal involvement. Some say that the appendix being a glandular organ like the tonsil, is liable to follicular infections, exudative and ulcerative.

Geo. F. Still states that the appendix vermiformis is a common habitat of the thread-worm in childhood, in fact he is strongly of opinion that it is the chief breeding-place for these worms. Their presence in the appendix causes a catarrhal condition with great swelling and thickening of the appendix, and very often there is severe pain which may stimulate ordinary appendicitis.

Foreign bodies, such as apple pips and other fruit seeds, bristles, pins, small shot, bits of bone, etc., are occasionally found. In 120 cases collected by the writer, only one had a foreign body, though 37 had enteroliths. In 174 cases collected by Archibald, only one foreign body occurred, and in one-third there were concretions.

Micro-organisms.—All the usual micro-organisms found in the intestines are seen here, the most constant being the bacillus coli, a normal inhabitant of the intestines. But the streptococcus pyogenes, staphylococcus pyogenes aureus, the proteus and bacillus pyocyaneus are also found—of course the most virulent is the streptococcus pyogenes.

No doubt at first all infection is mixed, and in the early stages of the disease the streptococcus has most to do with the production of the severe symptoms; bacteriological examination in all early stages will reveal the presence of both streptococcus and bacillus coli; later the temperature will fall and the colon bacillus will alone survive.

The colon bacillus is a normal inhabitant of the intestinal tract, and when a lesion of the mucosa occurs the organism gains entrance into the deeper tissues, and the more depressed the vitality of these tissues, the more virulently the bacilli act. They may spread by the lymphatic channels, by the blood itself, through the capillaries by penetrating their walls, and so lead to general septic infection. Streptococci act more locally, but the colon bacillus infects more widely, sometimes leading to a pylephlebitis and abscess of liver or lung, etc. There is now no doubt that when the colon bacilli collect in a blind pouch like the appendix, which probably has a stenosed exit, they become very virulent and cause attacks of inflammation.

Morbid Anatomy.—Every case of appendicitis is due to an infection from bacterial invasion of the tissues of the tube. The mucosa becomes eroded from pressure of enteroliths or concretions, and entozoa have been known to produce erosions. There may be an abrasion of the epithelial layer of the mucosa from violent muscular movements, sudden twisting of the appendix, or even direct violence. I have several times seen direct violence cause appendicitis, especially in those cases where a concretion existed. I saw one case in a child of ten years of age, which followed on a blow from a playmate; in this instance there was a large concretion. I operated on a lady for appendicitis, on whom the severe attack followed a blow from the handle of a bicycle; in this case there was no concretion. Anything which leads to an abrasion of the mucosa will tend to induce bacterial invasion and hence infection.

The severity of an attack of appendicitis is often determined by the virulence and extent of the infection and the resisting powers of the individual.

Anything which will produce an erosion of the mucosa will predispose to appendicitis. Micro-organisms may cause erosion or ulceration, but one of the chief causes of ulceration is the presence of enteroliths or fæcal concretions. Although in many cases these exist without causing any lesion, still, they are always a source of danger, and may produce an abrasion when subjected to pressure by severe muscular exertion or traumatism. Constrictions in the lumen of the tube may be due to the scars of old healed ulcers.

In the *Appendicitis obliterans* (Senn.), the so-called catarrhal appendicitis, which has already been referred to as a senile change, the mucosa is denuded of its epithelium and the muscular coats are much atrophied, the calibre of the tube is considerably reduced, and there is often injection and some slight adhesions of the peritoneal coat. The whole tube appears thicker, harder, and more resistant when palpated or felt with the fingers, and it seems to be in a state of erection. In the more advanced form, the mucosa is entirely destroyed and its place taken by connective tissue. The distal end is often completely obliterated, and converted into a solid mass of fibrous tissue. This process goes on at some points more rapidly than others and causes strictures; these can be easily shown by *Abbe's method* of distending removed appendices:—

Abbe's Method (*New York Med. Journ.*, July 10, 1897):—"Within a few hours after removal, the appendix should be distended with 95° of alcohol through the conical nozzle of a small syringe tied tightly into its cut end by a ligature, which is tightened as the syringe is withdrawn. The distended organ is then immersed twenty-four hours in alcohol of the same strength."

If it is now sliced centrally from end to end, its interior will be a revelation to the surgeon. He may find (1) a fæcal concretion blocking up the channel, (2) interior ulcerations, (3) cicatricial strictures, (4) multiple strictures with intermediate pockets containing suppurating and catarrhal products, and confined by greatly hypertrophied muscular and mucous coats, and (5) partial obliterating appendicitis.

In cases where there is a stenosis of the tube, colic is caused by the appendix endeavouring to extrude fæces and other matters which have happened to lodge in it. An abscess may form in the tube (Empyema), and by its rupture infect the surrounding peritoneum. Should the stricture be near the cæcal end of the process then it may dilate to a great size, looking like a sausage; if near the distal end the extremity often has a bulbous appearance. In some cases there is simply inflammation of the process, characterised by swollen mucosa and lymphoid tissue, with perhaps submucosal hæmorrhages and general thickening of the tube.

Results of the Infection of the Appendix by Micro-Organisms.

1. Chronic and Acute inflammatory conditions.

2. Ulceration and perforation as a result of the first condition, and leading to (*a*) abscess with limiting walls, or (*b*) general peritonitis.

3. Necrosis and sloughing—a more severe form of inflammation due to the excessive dose or virulence of the poison or the slight resisting powers of the individual.

Complicating these last two conditions we may have:—

1. Thrombosis of the ileo-colic vein with multiple or single abscesses in the liver.

2. Pylephlebitis.

3. Diffuse cellulitis of the retroperitoneal tissue.

4. A gangrenous inflammation of the cellular tissue and even of the neighbouring organs such as the kidney.

5. Thrombosis of the iliac veins with resulting œdema of the lower extremities, and occasionally pulmonary embolism, which of course is always fatal.

6. Cases of fatal hæmorrhages from ulceration into arteries, such as the iliac and circumflex, have been reported.

7. Suppuration of the mesenteric glands is also a complication which has been noted.

The SYMPTOMS of the various conditions are very difficult, and often impossible to differentiate; the mildest attack of appendicitis may suddenly assume a most serious character. Pain, tenderness, and slight fever will indicate in a general way an attack of the so-called "Catarrhal appendicitis," or simple inflammation of the appendix, but there may be no warning in cases of ulceration going on to perforation. The first symptoms may be sudden acute pain with vomiting, followed by rising temperature.

Continued high temperature, with rapid pulse and sometimes occasional chill, and the presence of a tender mass in the right iliac fossa with muscular rigidity or tenderness and resistance in the right lumbar region, would indicate the formation of pus.

Cases where *necrosis* and *sloughing* ensue often set in with great violence; an initial rigor, severe agonising pain, very rapid pulse and high temperature with continued vomiting (this often of a dark colour), would indicate sloughing. The whole appendix may slough and the gangrenous inflammation extend to neighbouring tissues and organs, but most commonly the sloughing is localised to one spot, at the tip of the process or along its free border; at one of these points there is often a fæcal concretion. Active processes leading to sloughing are now attributed to the presence of the colon bacillus, which under certain conditions becomes very virulent. In some cases thrombosis of the vein of the meso-appendix or the ileo-colic vein would predispose to gangrene. Morris attributes it to a proliferating endoarteritis of the single terminal artery; he also says that kinking or dislocation of the appendix will cause obstruction to the flow of blood in the vessels, and hence produce œdema of the process as in strangulated hernia, and this may go on to gangrene.

Perforation of the *appendix* is preceded by an adhesive inflammation of the peritoneum, which glues the coils of intestines together, does not infect the whole cavity, but a local suppurative peritonitis results, which has for its walls firmly adherent loops of small intestines and sometimes omentum. If the appendix be retro-cæcal (upwards towards the right kidney) it may become adherent to the posterior wall of the abdomen, and when the perforation takes place it is into the subperitoneal tissue. Should no attempt at localisation take place by adhesive inflammation, then the septic material is rapidly diffused into the whole abdominal cavity, and purulent peritonitis causes death in two or three days. The rupture of an abscess will produce the same result.

In many severe cases, suppuration does not take place, and inflammation may gradually disappear with the falling temperature. Most of us have seen cases where these large inflammatory masses have occurred again and again, always disappearing without going on to suppuration. I have operated in these cases, and found that the mass consisted, not of a loaded cæcum as many suppose, but of omentum, matted intestines, and inflammatory exudation with great thickening and enlargement of the appendix and neighbouring intestines. Usually a perforation in the appendix has not yet occurred, or if it has, at that point, there has been an attachment of the process to some neighbouring structures. In the 120 cases above alluded to, collected by the writer, there were 11 of gangrene and 36 with perforation.

The Formation and Results of Abscess.—In some cases in three or four days there may be quite a large amount of pus; even on the third day, I have seen abscesses of considerable size form. These cases where there is a localised tumour are comparatively safe, for the pus becomes walled off from the general peritoneal cavity. If evacuated before it bursts or extends to other parts, operation is usually safe. The continued increase of tenderness with continued elevation of temperature, would guide us as to the progress of the case and the necessity for operative interference. In some cases when abscess is present it is so small and hidden behind the cæcum that it is most difficult to find, it may be on the psoas muscle and connected with the perforated tip of the appendix.

An abscess may discharge into neighbouring hollow viscera, as the bladder, colon, and ureter. It is not uncommon in the female for pus to find its exit by the vagina, especially in cases where the abscess (secondary or primary) is in the pelvis.

There may be extensive extra-peritoneal suppuration, especially in cases where the perforation takes place into the retro-peritoneal tissue; pus may find its way below Poupart's ligament and may discharge here. Perinephritic abscesses often have their origin in the appendix, the pus may penetrate the diaphragm, get into the pleura and simulate empyema. Pus from an appendicular abscess has discharged itself through the obturator foramen and into the hip-joint, simulating hip-joint disease. The appendix has been passed by the rectum, and in one case related to the writer, it was coughed up, the abscess perforating the lung where it had become adherent to the diaphragm—until the appearance of the appendix, the medical attendant had never thought the case one of appendicitis.

Pylephlebitis has occurred which was only found out at the autopsy to be due to appendicitis, the symptoms being obscured by those indicating the existence of a pylephlebitis.

Symptoms.—An attack of appendicitis most commonly commences with a sudden and often very intense pain in the abdomen, followed in a short time by nausea, and perhaps vomiting, with tenderness in the right iliac region over the site of the appendix. There is a rising temperature and muscular rigidity on the right side of the abdomen—this is the picture of an ordinary onset of a case of appendicitis. If such a train of symptoms occurs in a young person, we may say the case is one of appendicitis, though we cannot tell what form of the disease the patient is suffering from.

Pain.—This usually sets in without previous warning, and is very severe, it very often comes on at night, and wakes the patient out of his sleep. At first it may be all over the abdomen, but specially in the vicinity of the umbilicus. Sooner or later it becomes localised to the region of the appendix, the point of especial tenderness being midway between the anterior superior spine of the ilium and the umbilicus (M'Burney's point). This pain has often been mistaken for biliary or renal colic, for the pain of indigestion or ordinary colic. The recurrence of sudden and severe pain without fever, called by Talamon "appendicular colic," is supposed to be caused by the endeavour of the appendix to get rid of fæcal contents or mucus in cases where there is a narrowing of its lumen. The appendix is thrown into violent peristalsis under these circumstances. The pain of a perforated tube, or of one in which gangrene is commencing, is often most agonising—in some cases of appendicitis an attack is preceded for some hours by moderate pain and discomfort in the abdomen, and later this becomes suddenly very acute. Constipation is the rule, though occasionally an attack may set in with diarrhœa.

Nausea and Vomiting.—In the great majority of cases, the pain is followed by nausea and perhaps vomiting; this is often very severe, but usually soon passes off after one or two retching fits. Vomiting, if continued, is a very serious symptom. When the vomited matter looks like coffee grounds the case is always unfavourable, for this is an evidence of general septic infection and of profound blood changes. These are the cases where under ether this vomit is apt to produce a septic pneumonia or bronchitis.

Fever.—Elevation of temperature soon follows the pain and is always present in the early stages of the disease. Except in cases of gangrene of the appendix, an initial rigor is rare, the fever in mild cases never runs above 102°, but in more severe cases reaches 104°-105°; in children it not unusually starts out with 102°-103·5°. A continually increasing temperature after the second day should give rise to grave apprehensions, and point to the necessity of considering the advisability of an early operation.

In some rare cases the temperature is normal; this is more likely to occur where there is a general purulent peritonitis; if with the low temperature there is a rapid pulse and respiration, immediate operation is indicated; a rapid pulse is always a serious symptom, that is, a pulse over 120.

Richardson of Boston states that in all cases of appendicitis with suppuration, if the blood be examined, leucocytosis will be found.

Local Conditions.—On inspection of the abdomen in the early stage, nothing abnormal is to be seen, but later, when septic paralysis of the bowels sets in, the abdomen becomes distended and the subcostal hollow becomes prominent and rounded, and there is general tympanites. The tenderness is always greatest at M'Burney's point, midway between the anterior superior spine and the umbilicus—very often there is great rigidity of the abdominal muscles on the right side; this is very characteristic in cases of perforation with sudden extravasation. Pressure over the region is very painful, and the tenderness often extends to other parts of the abdomen. If tenderness is felt in the right lumbar region, it is indicative of an inflamed appendix situated posteriorly having a direction upwards.

Tumour.—A distinct tumour cannot always be made out, but there may be a sense of resistance deep down or an ill-defined induration in the right iliac region. If a tumour forms, it is well-defined, hard, and situated in the neighbourhood of a line drawn from the umbilicus to the anterior superior spine of the ilium. In many cases no tumour forms at all; these are the cases where a perforation occurs without any previous limiting inflammatory area, and the contents of the bowel are emptied into the general peritoneal cavity, giving rise to a most virulent form of peritonitis. In many cases the trouble is limited to the interior of the appendix at first, and there is no external evidence of a tumour. In all long-continued cases we are likely to have a tumour mass—there are cases where a tumour may exist, and yet it is situated too deeply to be distinctly made out.

Position.—In most cases of appendicitis the dorsal decubitus with the right leg drawn up is the most comfortable position; the patient will tell you that he is unable, on account of the increase of the dragging pain, to lie on his left side.

Rectal Examination.—It is always well to make an examination of the pelvis through the rectum, for if the appendix hang over the brim of the pelvis one can always get tenderness, and if there be an abscess in the pelvis it can always be felt by rectal examination. When the appendix is inflamed and in the pelvis, painful micturition, retention, and tenesmus are apt to occur. In most cases, however, nothing can be made out early by a rectal examination.

Palpation of the Appendix.—It is possible in early cases, and cases of chronic and recurrent appendicitis, to palpate the appendix; this has been pointed out by Dr. Geo. Edebols of New York. Many assert they can recognise the appendix even when healthy—when enlarged and thickened, they have no difficulty in identifying it.

PROGRESS OF THE CASE.—In the majority of cases recovery is the rule, though some of these may succumb to a subsequent attack. A large number go on to the formation of abscess, which if not relieved may rupture and cause a general peritonitis. A certain number develop a general peritonitis almost from the first (Fulminating).

In cases which tend to recover, the tenderness and pain begin to diminish at the end of the second day, the temperature also falls and vomiting ceases early. There may be tenderness, pain, and some fever for a week or more, but the case gradually gets better and the countenance assumes a less anxious look.

A tumour may persist for some time, but this gradually gets smaller and less tender until finally it disappears altogether. Subsequent attacks in such cases are characterised by the reappearance of the tumour.

Very often there is no tumour to be felt in the recurrent cases, nothing but the initial vomiting, severe and sudden pain with muscular rigidity, to indicate the attack; in such cases the inflammation is confined to the appendix itself and to the peritoneum covering it.

Should the case not be relieved by operation, and go on to general peritonitis, the pain which was confined to the right iliac fossa now extends to the whole abdomen, the muscles of the abdomen become very rigid, vomiting continues unabated, the vomited matter being fæcal in odour and yellow or dark in colour; there is paralysis of the bowels, the patient passes neither flatus nor fæces, and the abdomen becomes much distended; in some cases the meteorism is not marked; there is retention of urine. As a rule the temperature is not high, it may even be subnormal; the pulse is small and rapid, and the surface of the body is cold and sweating. The patient has the typical abdominal face, anxious shrunken features, pinched nose and sunken eyes; the voice becomes weak, the tongue dry and red—and the patient dies in three or four days from exhaustion.

General abdominal distension is always a serious symptom in appendicitis and is evidence of commencing paralysis of the bowels and perhaps general peritonitis, but if there is any evidence of peristalsis, if borborygmus occurs, there is usually no general peritonitis, and the outlook is favourable.

In the so-called fulminating cases, where the peritonitis is generalised from the first, the symptoms set in most severely. Intense pain, very often an initial rigor, great rigidity of the abdominal muscles, temperature not much elevated, but the pulse is small and rapid? The abdominal face early appears and the patient is evidently collapsed—vomiting is persistent and of a dark colour—patient usually dies in two or three days. In such cases, immediate operation may occasionally save a life.

Recurrent or Relapsing Appendicitis.—When a person has had two or more attacks of appendicitis, we speak of it as "recurrent or relapsing appendicitis"—usually these attacks are never very severe, though they have the ordinary symptoms, such as sudden and severe abdominal pain which soon becomes localised to the right iliac region, the point of greatest tenderness being at M'Burney's point, nausea or vomiting and elevation of temperature with rapid pulse. Not infrequently a tumour forms in the right iliac fossa, which disappears with the subsidence of the pain, tenderness, and fever, to reappear again at the next attack.

In some cases months may elapse before another attack comes on, and in the interval the patient is perfectly well—these are the true *recurrent cases.* In other cases the attacks are much more frequent, every few weeks, and between attacks the patient is out of sorts, has constipation or diarrhœa, digestive troubles, flatulence and occasional colicky pains, pain on pressure over M'Burney's point, and this pain is increased by exercise and over-exertion of any kind. In some cases the patient is unable to lie on his left side without a sense of dragging pain, in these cases the appendix may often be felt—this is the true *relapsing* or *chronic appendicitis.* It is stated by Fitz that 11 per cent of all cases are of the recurrent or relapsing type, others place it much higher, 22–23 per cent (Krafft, Hawkins). In the cases collected by the writer, over 50 per cent had had previous attacks.

Diagnosis.—Appendicitis is the most common inflammatory affection of the right side of the abdomen. It is the rare exception that peritonitis in the male is due to anything else than appendicitis. According to Krafft only 2 per cent of peritoneal inflammations in the male are not due to appendicitis. The exceptions being volvulus, perforation in other parts of the intestine, *e.g.* duodenum, stomach, etc., intussusceptions, contriction by band, twists, mesenteric thrombus, etc. Einhorn reports that in a hundred deaths from perityphlitis, ninety-one had their origin in the appendix, nine were due to perforation of cæcum from stricture.

Any affection which sets in with sudden and severe abdominal pain, shortly afterwards localised to the right iliac fossa, with fever and tenderness over the region of the appendix, with or without nausea and vomiting, is most likely to be appendicitis.

Typhoid fever has been mistaken for appendicitis. Typhoid fever is preceded by malaise, there is no sudden severe pain, and the tenderness is not so marked in the right iliac fossa. In persons who have had previous attacks of appendicitis, the diagnosis may be difficult, and cases of atypical typhoid fever have been operated on for appendicitis. Abscesses in connection with the cæcum after typhoid fever cannot be diagnosed from abscess following upon appendix disease. Perforation from typhoid ulcer occurs after the disease has lasted some time and typhoid would have been recognised by the blood reaction, eruption, etc.

Biliary and Renal Colic.—This may be mistaken for appendicitis. In biliary colic the pain is nearer the epigastrium and vomiting is a feature; there is often tenderness beneath the costal border, and history of previous attacks and jaundice. In renal colic the pain radiates down the ureters to testicle, and there may be tenderness over the kidney. In neither is there fever.

An *inflamed and distended gall-bladder* may simulate appendicitis. The writer operated on such a case; patient was seized with sudden and severe pain, tenderness in right iliac region, fever and vomiting, and a tumour was found in a line between the umbilicus and anterior superior spine. On opening the abdomen in the usual way an inflamed distended gall-bladder presented itself which was opened and some stones removed, and the patient recovered rapidly.

Neuralgia of the lumbo-abdominal muscles has been mistaken for appendicitis, as has also coxalgia. The paresis of the bowels which accompanies a severe case of appendicitis has been frequently mistaken for intestinal obstruction due to twists or bands; here, however, temperature is a late feature of the case. I have seen cases of localised gangrene of cæcum from cancerous disease blocking the mesenteric vessels simulate appendicitis closely, and cancer of cæcum has been mistaken for the chronic form of appendicitis. In some cases where an abscess has followed perforation, the diagnosis is impossible without exploratory incision. As has been before said, appendicitis may be the cause of perinephritic abscess, empyema, psoas abscess, etc.

Osler mentions that mucous colitis with enteralgia in nervous women is sometimes mistaken for appendicitis. He also says that there is in America a well-marked "*appendicular hypochondriasis*," and such has been my experience.

In the female, *pelvic inflammation* may be mistaken for appendicitis and *vice versa*. In some cases the appendix is involved with the tubes. When the inflamed appendix hangs over the brim of the pelvis it may be mistaken for pyosalpinx, pelvic hæmatocele, or again pyosalpinx may be mistaken for appendicitis. In cases of pelvic inflammation, rigidity of the abdominal muscles is always absent.

Duodenal ulcer has been mistaken for appendicitis, especially when it has perforated. In such cases there may have been previous passages of blood per anum, or blood may have been vomited. It is always well to examine the region of the duodenum in cases where at operation the appendix is apparently healthy. Perforating ulcer of the stomach may simulate appendicitis.

Prognosis.—No doubt a great many cases of simple appendicitis recover, and every practitioner has seen cases where attacks have occurred several times. Hawkins states that in 154 cases treated at St. Thomas' Hospital in which there was no suppuration, only one died; Morris places the death-rate in unoperated cases at 25 per cent. It is very difficult to get any satisfactory statistics as to the mortality of cases which have not been operated upon. From the appearance of the appendix in ordinary post-mortems, quite a large proportion show traces of former inflammation; many cases are cured spontaneously by the separation of the appendix and its discharge with the surrounding abscess into some neighbouring hollow organ, most commonly the cæcum or colon. I have seen quite a number of such cases.

The prognosis after operation in the interval is always good. Richardson of Boston reports 151 cases without a death. W. T. Bull's statistics give 412 cases with a death-rate of 1·8 per cent. In cases operated on during the acute stage, the prognosis depends on the period at which the operation takes place, the character of the disease, and the skill of the operator. Probably the mortality after operation in all acute cases admitted into hospital is about 15-20 per cent. The prognosis in cases after operation is always good when the inflammation is localised, and it is always an encouraging symptom if after operation the bowels move freely.

Surgeons who see only the most severe cases are very apt to have false ideas of the great mortality in cases of appendicitis which are not operated on.

Treatment.—This might be divided into *Expectant and Operative. Expectant:* In ordinary cases the patient should be kept in bed on milk diet and the course of events watched—medicine will do but little for him, though such drugs as calomel, salines, opium, etc. are advocated as curative remedies by enthusiastic therapeutists. To relieve the pain, applications of heat by means of fomentations, or cold by means of ice-bag, are most acceptable to the patient—personally, I prefer the ice-bag; not only does it relieve the pain, but, should operation be afterwards undertaken, there is less congestion of the superficial parts and the skin remains intact; nothing is so disagreeable as operating on a patient whose abdomen is raw from the frequent application of counter-irritants, or blistered from continuous applications of too great heat. In such cases it is impossible to cleanse the field of operation, and the risk of sepsis is much increased.

Purgatives should be avoided, as in the early stages they may do considerable harm, especially if there be a perforation with a commencing localised adhesive inflammation. The cæcum, as was stated in the beginning of this article, is nearly always empty and never loaded with fæces, so emptying it would be of no service. If the pain be very severe, small doses of opium may be given, but opium is a dangerous friend in appendicitis, and is very apt to conceal serious symptoms and lull the suspicions of the attending physician. If at the end of forty-eight hours the case does not improve, that is, should the vomiting not cease, the temperature continue to rise and the pulse-rate be over 120, with increasing rigidity of the muscles and anxious expression of face, then the question of operation should be seriously entertained. It is often a difficult matter to know when to operate—some surgeons, as Morris of New York and Murphy of Chicago, say that whenever a case is recognised as one of appendicitis, operate. But all surgeons are not prepared to follow this advice; it is certainly a solution of the problem, but it is not a necessary one in all cases.

Should the symptoms not ameliorate, the pulse remain rapid, local tenderness and muscular rigidity increase, or should a distinct tumour develop and the general appearance of the patient be alarming, then operate—in such cases one is very likely to find pus. Immediate operation is indicated if there is a rapid pulse and respirations with a normal temperature.

Gay of Boston says one should not operate on a patient in whom the symptoms are rapidly subsiding; if operation is desirable, it should, in such a case, be undertaken a week or ten days after all active symptoms have disappeared.

A person who has had two attacks of appendicitis had better have the offending organ removed, for he is liable to other attacks at any time, and possibly the next attack may be a severe one, and perhaps perforation may take place. The fear of an attack keeps the patient in continual dread; he is unwilling to leave home, dreading he may have an attack when out of reach of skilled surgical assistance. It must be confessed, that in many cases, repeated attacks take place and then they cease and the patient gets perfectly well, but one can never tell these cases from those which may suddenly develop a perforative peritonitis with its subsequent dangers. Operation should be advised after a single severe attack, if the patient does not entirely recover, or if a tender appendix or tumour remain after the subsidence of the fever.

In cases of recurrent appendicitis, attacks may frequently be avoided by careful attention to diet and regular habits. Constipation above all things should be avoided, also late suppers and very hearty meals—all indigestible food should be carefully shunned. An occasional dose of some saline purge is beneficial. Exposure to cold should also be avoided, also over-exertion. In many cases, with the most careful diet and the taking of every other precaution possible, the patient will still have attacks. In these circumstances nothing but operation will offer any relief from the recurring trouble

OPERATION: DURING THE INTERVAL.—Operation during the interval may be undertaken with great confidence. The mortality in all cases in the hands of skilled surgeons is about 1 per cent. The best time to operate is about two weeks after the attack, or as soon as the acute symptoms have subsided. After cleansing the field of operation and the hands of the operator, and taking other precautions which are necessary in every important surgical operation, an incision should be made, as recommended by M'Burney, crossing an imaginary line drawn from the umbilicus to the anterior superior spine of the ilium at the junction of its middle and lower thirds; this incision should be downwards and inwards in the line of the aponeurotic fibres of the external abdominal oblique muscle one and a half inches internal to the anterior superior spine of the ilium. The external incision should be from 3″ to 4″ long according to the amount of adipose tissue; only the skin and aponeurosis is incised; the fibres of the external abdominal oblique are separated and held aside by an assistant with blunt hook retractors; the internal oblique is now exposed and its fibres are seen running at right angles to the first incision; the fibres of this muscle and of the transversalis are now separated without cutting by means of the handle of a scalpel, another assistant with another pair of retractors holds these muscles aside. After all hæmorrhage has been arrested and the hands washed freshly, the transversalis fascia and the parietal peritoneum are cut through in the same direction in which the deeper muscular fibres have been separated. These structures are also held aside with the deep muscles, and the peritoneal cavity is now seen with some bowel or omentum presenting; the bowel is usually the cæcum or colon, and the anterior muscular band must now be looked for—when found it is traced down until the appendix is reached. The appendix may often be found by introducing the forefinger into the wound, and if there are no adhesions it may easily be turned out through the wound.

It is sometimes necessary to draw a small part of the cæcum out of the wound to enable the operator to reach the appendix; it often springs up into the wound if there are no adhesions, and then is easily isolated and removed. When it is reached, if necessary, free the adhesions about it and then tie off its mesentery by passing a fine silk or catgut ligature through the mesentery near the cæcal end of the appendix. Having tied and cut this away, the appendix is free—the distal end should be seized and a ligature of fine silk or catgut placed about it near the cæcal end, and the appendix cut off with scissors or burnt through with a thermo-cautery; however divided, it should be carefully protected during the process and after division—a sharp point of a well-heated thermo-cautery should sear the cut surface and also the exposed mucous surface on the distal side of the ligature. The heated point should be pressed deeply in so that the infected mucous membrane will be thoroughly destroyed.

Some prefer the applications of pure carbolic acid to using the cautery. It probably answers equally well. After the stump of the appendix has been thoroughly disinfected, it, with the cæcum, is dropped back into the peritoneal cavity and the deep retractors removed; the wound closes almost of itself—in the deep muscles it is not more than 2″ long. The divided structures are now sutured separately with catgut, a continuous suture being used. First the peritoneum and transversalis fascia, next the internal oblique and transversalis, then the aponeurosis of the external abdominal oblique, and finally the skin. It is my custom to use interrupted horse-hair sutures for the skin incision. No drainage is used as a rule, but should there be any pus, which in interval cases is rare, a small drain of sterilised cotton-wick might be used for twenty-four hours.

Of course this operation was never intended or recommended by M'Burney in suppurative cases. In these cases the ordinary straight incision through the various structures forming the wall of the abdomen should be made use of. I have now used M'Burney's incision, which is called the "criss-cross" or "grid-iron" incision, in a great many cases, and have never yet seen a hernia result. It makes a perfectly safe wound, because at only the point of crossing of the two incisions is there any through communication between the skin and peritoneal cavity—no nerves or muscles are cut and no anæsthesia follows. It must be confessed in cases where more room is needed owing to difficulties which have arisen, the incision has to be enlarged into a straight one, so that an ugly wound is the result; but the necessity for changing the incision in ordinary interval cases will not often arise. I have had to change the incision only once in two years; the case did perfectly well, and no hernia resulted. Another objection which has been urged against this operation is the number of assistants required.

Many other incisions are recommended—the oblique incision inside the anterior superior spine, beginning a little above the line drawn from the anterior superior spine to the umbilicus; of course such an incision may be enlarged up and down as required.

After-Treatment.—The patient should be kept in bed and perfectly quiet for at least three weeks. For twelve hours after operation nothing should be taken into the stomach but occasional sips of hot water—an enema of soap-suds and turpentine (℥j-Oj) should be administered on the second day; this usually gives great relief. If this is not effectual, salines should be given by the mouth. Liquid food may be taken on the second day, peptonised milk, or milk and lime-water, and afterwards milk, toast, etc., till finally, after the first week, the patient can eat almost anything. The dressing in these cases need not be removed till the sixth or seventh day, when stitches are removed.

In the interval cases convalescence is rapid and uninterrupted. I never advise the wearing of a truss afterwards if the wound heals without suppuration.

Operation in Acute Cases.—The indications for operation in acute cases have already been given. It is always better to operate if one is in doubt, for early operations are comparatively safe, I have never repented having operated too early, but have been sorry in many cases in my early experience that operation had been postponed to a period when it was too late.

In acute cases, when we cannot tell beforehand whether or not pus is present, a direct incision through the abdominal walls is to be preferred. The incision should be made outside the edge of the right rectus muscle, commencing almost an inch above a line drawn from the umbilicus to the anterior superior spine of the ilium; the incision should be parallel to Poupart's ligament, and about two inches internal to the anterior superior spine. Of course if there be a tumour, the position of the incision may be changed to the most prominent part of the tumour. The writer has frequently opened the abdomen in the middle line with benefit on account of the situation of the tumour being more central than usual. In some cases also where the inflammatory swelling is higher up and more posterior, an incision is made more above the anterior superior spine than below it.

It is always well at first to make a small incision, having its centre opposite the spine, and then extend it upwards or downwards as occasion may require. Having cut through the abdominal wall and come down on the transversalis fascia and peritoneum, these should be divided, and now the inflammatory area, of which the appendix is the centre, should be searched for; but first, the general peritoneal cavity should be carefully protected by the introduction of sterilised gauze pads between the parietal peritoneum along the edge of the wound and that covering the intestines. When this has been carefully done, the inflammatory area should be examined; the adhesions, if operation has been performed early, are usually soft and easily separated, and soon a gush of stinking or sero-pus is seen welling up into the wound; this should be swabbed out with gauzes as soon as it appears and until it ceases—the gauzes of course should not be used again without being sterilised. Now the appendix is searched for, and is commonly seen lying in the abscess cavity; it is usually enlarged, red, and inflamed, with a perforation, and perhaps gangrenous. The appendix can be easily separated with the finger, for the adhesions in which it is embedded are usually soft. It should be traced up to its attachment to the colon, and its mesentery should be tied off with a single ligature if not too broad—in sections, if broad. A ligature should now be placed around the appendix itself, one-eighth to one-quarter of an inch from the cæcum, and treated as described in the interval operation.

M'Burney recommends that the appendix should be cut off before a ligature is applied, and that a probe should always be passed into the cæcum to show the patency of the stump, for in some cases he has seen an impervious stricture between the section and the cavity of the colon.

In some cases there will be no perforation or gangrene and no pus, the inflamed appendix will easily be turned out of its bed of soft and easily broken down adhesions; in other cases again the distal end is so embedded in old inflammatory tissue that it cannot easily be shelled out with the finger—in such a case the cæcal end should first be freed, and then the appendix dissected down from there to its apex.

In some cases, again, the appendix is attached to intestine at the point of an old perforation, and the separation has to be undertaken with the greatest care, for a tear may be easily made in the bowel at the point of attachment. It is sometimes well to leave a portion of wall of the appendix attached to the bowel. Not infrequently it is very difficult to find the appendix, it is so hidden away in adhesions; but the greater one's experience, the fewer appendices are left behind—one's tactile sense becomes so educated that the finger after a time can easily detect an altered appendix though it may be quite out of sight.

It is always better to remove an appendix if its removal does not involve too much dissection or too much disturbance of the limiting wall which separates it from the general peritoneal cavity. In cases of gangrenous appendix the gangrene may extend so far that it is impossible to tie off the appendix from the cæcum; in these cases the stump is so short that the ligature holds only insecurely, and the condition of the tissue forbids suture. In such cases inversion of the stump into the cæcum has been suggested, and then the inverted part closed over by Lembert's sutures.

It not infrequently happens that on opening the peritoneal cavity the thick wall of the abscess cavity is seen on the posterior wall. In such cases the general cavity should be carefully separated before opening it by aseptic gauze pads, as described above, then the abscess opened with the patient turned on his right side. As soon as pus appears, it, of course, must be swabbed up with gauzes.

Again, the abscess cavity may be surrounded by coils of intestine weakly held together by soft adhesions. In these cases great care must be taken not to infect the general cavity, and careful keeping away of the intestines by the assistant's hands or pads must necessarily be attended to. Operation in acute cases of appendicitis may be very simple or very difficult, and only experience will tell us how to most easily overcome the difficulties.

The simple cases are those when on cutting through the abdominal wall we find it œdematous and infiltrated. We know that the abscess is near the anterior wall, and, in fact, it may be attached to it, so that on opening the abscess there is no need at all of opening the peritoneal cavity. In all cases it is better to keep out the pus with sponges or gauzes than to wash it out; there is much less chance of its being disseminated. Many surgeons are accustomed, after the pus has been removed and the appendix tied off, to wash out the cavity with saline solution, but the writer has given up this custom for some time and finds his results equally good. Some surgeons also carefully peel off the lymph which covers the intestines; this is quite unnecessary and may be dangerous because of opening up fresh avenues of infection. According to Welch of Baltimore, this exudate is always sterile.

Now, after the abscess cavity has been made as clean as possible and all hæmorrhage arrested, it should be packed with iodoform or sterilised gauze, the wound partially closed—how much, depends on the judgment of the operator. It is the writer's custom to suture the wound in layers, leaving a part below unsutured through which the gauze comes out. Should the abscess extend behind the colon, it might be as well to put a drainage tube through the wound and out at the flank. This is easily done by thrusting a pair of artery forceps through the soft tissues of the posterior wall and cutting down on them through the skin; when they are thrust through the skin the incision may be enlarged slightly and the blades of the forceps opened, and into them put a drainage tube, which is withdrawn with the forceps. This method of posterior drainage the writer has found most useful in these cases; the drain need not be left in long, an anterior drain is of little use. Some advise cotton wick where a small drain only is needed; a strip of green protective silk tissue is very useful in such cases.

When the diseased appendix is situated near the brim of the pelvis, or hanging over the brim, a pelvic abscess not infrequently results. It is well in all cases where the appendix is low down to explore the pelvis before closing the wound, and if pus be present to insert a glass drain with gauze packing about it, through which pus can be sucked at intervals with a glass syringe and rubber tube attached which can go inside the glass tube; a strip of gauze in the tube acts well as a drain.

Secondary abscesses not infrequently occur here. It is possible sometimes to open these abscesses through the rectum, and so get dependent drainage.

After suturing, dry sterilised gauze should be placed next the wound, and over this a large mass of aseptic absorbent cotton wool, and over all a bandage or a couple of broad strips of rubber adhesive plaster.

The outer dressings should be changed in twenty-four hours, and will be found soaked in serum; the packing should be left twenty-four hours longer, and then removed with great care—this procedure is often quite painful, and in children an anæsthetic may have to be administered. After cleansing the wound with pledgets of sterilised cotton, it should be repacked, but not so firmly; of late I have used strips of sterilised gauze in place of iodoform gauze, and find it is easier to remove and more absorbent. The dressing is renewed every two days, less gauze introduced every time, until it is left out altogether, and the rest of the wound heals rapidly.

After operation the bowels are frequently distended with gas, and to relieve this painful condition an enema of soapsuds and turpentine (℥j.-Oj.) should be given during the first twenty-four hours; this greatly relieves the patient. If the bowels do not move after forty-eight hours the patient should be given half a grain of calomel every hour for six hours, or five grains at one time. This, followed by Rochelle salts, is very effective. Calomel can easily be taken, even when the patient has a sick stomach. In some cases salines act very effectively, but in others the patient cannot retain them. In one apparently hopeless case, in which the writer operated, where there had been free pus in the abdomen and a gangrenous appendix, and where the operation was performed on the fifth day as a forlorn hope, the bowels, which were apparently paralysed by sepsis, could not be got to act by any method or the use of any drug. At the request of the patient, a medical man, half a grain or more of morphia was injected hypodermically to relieve pain, and it not only did this, but seemed to stimulate the bowels, so that peristalsis was excited and a free movement resulted. The patient made an excellent, though slow, recovery.

In the cases in which operation has been performed at a late period, or where there has never been any localised exudate, to protect the peritoneum from the results of perforation and general peritonitis, the treatment must be somewhat different. After the abdomen has been opened by a free incision and the diseased appendix removed, the patient should be turned on his side and as much pus as possible allowed to escape; the abdomen should then be flushed copiously with hot normal saline solution (℥j.-Oj.), and all pus washed out—when the solution comes away clear the flushing may be discontinued. Some of the solution may be left in the abdomen, for it acts as a stimulus to the heart and promotes intestinal drainage. During the washing out the irrigating tube must be thrust into all the holes and corners of the abdomen. Sterile gauze should now be introduced in all directions, also down into the pelvis, to the stump, and up the right flank. The ends of the strips of gauze should be brought out of the lower end of the wound, which has been only partially closed.

When the patient is leaving the operating table $\frac{1}{4}$ gr. of morphia, with $\frac{1}{100}$ gr. of atropine, may be given hypodermically, and stimulating and nutritious enemata frequently administered. It is also very important that the bowels should be got to act as soon as possible; the best remedy in these cases is calomel.

There are many other methods made use of in such cases. Some recommend the taking out of the intestines and washing them by pouring hot saline solution over them. Others get an assistant to hold up the edges of the wound, and then the abdomen is filled with the saline solution; and the operator, with one hand holding the flushing tube, stirs the bowels about with the other, being careful to wash out the pelvis, lesser sac, the recesses about the liver, spleen, and other places. M'Cosh advocates in these cases "the injection into the small intestines of an ounce of magnesium sulphate in a saturated solution, the needle puncture being generally closed by a Lembert suture," also the administration of ten grains of calomel as soon as the patient recovers from the anæsthetic.

In cases where vomiting persists, washing out the stomach is often of great service. M'Cosh reports nine cases of general peritonitis, with three recoveries, treated by his method. In cases of general peritonitis, the earlier the operation the better the results; though all the cases are desperate, yet by the means mentioned above an occasional one may be saved.

Methods of treating the Stump.—Very many methods have been devised for removing the stump, some simple and some elaborate—the simpler the method the better. Tying off with catgut and removing with scissors, then cauterising the cut surface and lumen freely with the fine point of a thermo-cautery, has given in my hands as good results as any of the more elaborate methods, all of which have been tried and abandoned; methods such as inverting the stump into the cæcum, and then closing with Lembert sutures, making a cuff of peritoneum and covering the end of the stump, invaginating the stump and closing the opening with a continuous purse-string suture as recommended by Dawbarn, and other methods without number. All the elaborate methods take time, and give no better results than the simpler method mentioned above.

APPENDICITIS COMPLICATING PREGNANCY

In these the attack begins with sudden and severe abdominal pain, which afterwards becomes localised, vomiting, and fever. It may be confounded with extra-uterine pregnancy. When complicated with pregnancy the prognosis is always grave.

FÆCAL FISTULA

A fistula may persist from perforation of the cæcum or gangrene of the cæcal end of the appendix. It may also be caused by a fæcal concretion which has escaped and remains behind. Most of the cases heal if they are left alone and kept clean. Should a concretion be the cause, it must be removed. Operation for fæcal fistula should not be undertaken too soon, and when it is performed the abdomen should be opened away from the fistulous opening.

HERNIA OF THE APPENDIX

In cases of cæcal hernia the appendix is always present. The writer operated on a case of strangulated inguinal hernia some years ago where there was intense pain and vomiting, and with the herniated small intestine was the appendix tightly constricted. It occasionally happens that the appendix may be the only portion of the bowel in the hernial sac.

ACTINOMYCOSIS OF THE APPENDIX

This parasite may enter any part of the intestine, but usually prefers the appendix. It may penetrate its walls, cause suppuration, ulceration, and necrosis. The inflammation may spread to the peritoneum and subperitoneal tissue. It is different from ordinary appendicitis, because it is of slower progress and the symptoms are less acute. It is practically painless. A great deal of infiltration takes place, and numerous fistulous tracks are formed; there is no involvement of glands and the growth remains very circumscribed (Jamon). It is a very rare disease.

Tuberculosis of the Appendix

This is not a very common affection, it cannot be distinguished from the ordinary cases until removed and examined. The writer removed a tuberculous appendix from a man aged thirty-five who had had repeated attacks of appendicitis. Six months later he had general peritoneal tuberculosis, which proved fatal.

Typhoid ulcers may occur in the appendix, and *cancerous* disease may involve it secondarily from the cæcum or ileum.

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Appetite.—The desire for food depends ultimately upon tissue metabolism, a mechanism by which nature repairs the body waste. External cold, or a cold climate, by promoting muscular activity, increases metabolism, and with it the desire for food, while the converse conditions lead to the opposite result. Under pathological conditions increased metabolism may create no demand for food, owing to the intervention of other factors, and even in health this desire is subject to many influences which modify it in various ways. Civilised adults, accustomed to a regular supply of food at fixed hours, usually manifest a desire for food only at those times: in other words, their appetite has become a habit; but children and savages are always ready to eat. The smell or sight of food, or even the effect of imagination, may awaken the desire, while predilections of taste undoubtedly stimulate appetite, certain articles of food proving irresistible to many persons. On the other hand, mental preoccupation, surprise, or shock may hinder the perception of the desire for food, and habitual meal-times may thus pass unnoticed.

Loss of Appetite is the common effect of any debilitating or depressing influence, although there are exceptions to this rule, of which the well-known excessive hunger of diabetes is perhaps the most striking. In all fevers and acute infectious processes appetite is diminished or lost, in spite of increased metabolism. This loss of appetite has been by some attributed to the reduction of the digestive functions of the stomach; but the most probable explanation is that the morbid poison depresses all the vital activities of the body, including the desire for food.

In acute and chronic gastritis, where nausea is present, there may be temporary loss of appetite or distaste for food, amounting to loathing; but this is exceptional, and as a rule those patients who suffer from such affections of the stomach require to have their appetite controlled. Persistent distaste for food in chronic stomach disease is in fact a highly significant symptom of cancer of this organ; but such a remark must be limited to the differentiation of a certain group of cases, viz. chronic dyspepsia in elderly persons. Total loss of appetite accompanied by pain after food and persistent vomiting is met with in a purely functional nervous affection, anorexia nervosa.

The desire for food may be temporarily abolished by the use of alcohol, tea, tobacco, opium, cocaine, and other drugs, while the abuse of any of them may result in total loss of appetite. These effects vary with the individual and the dose, many persons being able to enjoy food while indulging in considerable quantities of one or other of these substances.

The treatment of loss of appetite consists in the removal of the causes as far as they are removable, especially in the improvement of the general health by the disuse of nerve poisons such as alcohol, tea, tobacco, opium, etc., and the increase of metabolism by the use of such remedies as change of air, mountain and sea air, cold baths, douches, massage, exercise, riding, cycling, walking, and the use of bitter tonics, for example, quinine, strychnine, quassia, calumba, and gentian.

INCREASE OF APPETITE.—The desire for food is increased, as already mentioned, when metabolism is hastened, and, although in acute infectious processes this relation, during the height of the fever process, is obscured, as convalescence sets in appetite returns with all the energy needed to make good the loss that has taken place. In diabetes, as a rule, and less constantly in Graves' disease, there is an excessive craving for food.

Unfortunately the appetite is too often great when metabolism is normal, and the intake of food is then largely in excess of the needs of the body. The causes of this unnatural hunger, or *bulimia*, are by no means clear. In children and others such a craving is sometimes associated with the presence of intestinal worms; in women, with pregnancy and hysteria; in both sexes, with gout, epilepsy, neurasthenia, insanity, cerebral tumours, etc.

Observation shows that there are at least two elements in these affections—(*a*) over-stimulation of the desire for food; and (*b*) absence or diminution of the sense of satiety.

Some may doubt whether there is a sense of satiety, as distinct from the satisfaction of the desire for food. The writer believes that in the gouty form of bulimia there is this absence of a sense of satiety in spite of the ingestion of enormous quantities of food, and long after the desire for food has been appeased, for if these patients have the amount of food limited they suffer no discomfort. In neurasthenia, on the other hand, although there is frequent complaint of emptiness and craving, this feeling is very soon satisfied and the quantity of food taken at one time is probably little if at all excessive.

Excessive desire for food is probably the general rule in bulimia of purely nervous origin, although both factors may co-operate. Undoubtedly savages, like the wild carnivora, gorge themselves whenever they have an opportunity, because they are unable to reckon with certainty upon their next meal, and the habit of eating enormous quantities survives the need for it. Among children, and in demented and imbecile persons, there is a reversion to this condition, so that it is often necessary for those who have charge of them to regulate their supply of food.

During recent years the digestive functions of the stomach have been in innumerable cases carefully determined, and it is certain that there is no close correspondence between the depression of these functions and the state of the appetite. In order to elucidate this point further the writer addressed to Dr. Schlatter of Zurich, whose remarkable case of complete excision of the stomach (*Lancet*, 1898, vol. i. p. 141) will be within every one's recollection, an inquiry as to the persistence of 'appetite' or 'desire for food' in his patient. His reply, dated January 23, 1899, in the original English, is as follows:—'It is with pleasure I give you any information in my power on the subject you wish. I say in my second report on the case (*Lancet*, Nov. 19, 1898, p. 1315), "Until a short time ago she asserted that she had quite lost the feeling of hunger through the operation, but, etc." I asked the patient several times if she felt hungry, and received the reply that she never had the same feeling of hunger as before. But it was with some astonishment I noticed that she frequently asked for food, about every three hours. When I inquired of her why she asked for food when she was not hungry she only replied, "Mankind must eat." Therefore I cannot say with any certainty that the craving for food had quite left her, but *she had the impression that such was the case.*'"

This interesting letter leaves the question unsettled, although it gives some support to the opinion that the desire for food, *so far as it is a sensation*, originates in the stomach itself.

PERVERSION OF APPETITE—*Pica.*—The conditions, which may be described under the head of perverted appetite, or pica, vary very much in degree, and may be considered to include—(1) dislike of wholesome food; (2) liking for unwholesome food; (3) liking for substances unfit for food, improper, or disgusting.

(1) *The first of these conditions* is very commonly met with in children, whose range of palate is often at first so limited that they will only eat the food with which they are familiar. Dislike of meat is common among them, even of lean meat, although it still more frequently takes the form of aversion to fat.

This sort of capricious appetite is by no means unknown among adults, especially among women, who retain throughout life some of the characteristics of childhood.

(2) *The liking for unwholesome food* is a perversion of appetite which is not only within normal limits, but is probably met with among the majority of mankind. The fondness for putrefying flesh, cheese, and some kinds of fruit; for indigestible solid puddings, for greasy cookery, for oily fish, such as salmon, eels, lampreys; for shellfish, sweetstuff, etc., can only be explained by the acceptation of the view that the appetite is easily perverted by the sense of taste, and that the latter is not a safe guide in the choice of food.

(3) *In the third category*, however, we pass without doubt beyond the limits of the normal and enter the domain of pathology. The substances eaten are chalk, plaster, slate-pencil, coal, cinders, hair, paper, wood, filth, fæces, etc.

In pregnancy there is often a capricious appetite, not seldom an overwhelming desire for particular food which may or may not be wholesome, and sometimes a disposition to nibble at chalk, plaster, etc. In hysteria the same aberrations occur, but have a wider range, extending to probably all the substances enumerated, although usually it is only among the truly insane, idiotic, or demented that stercophagy is met with.

Arcus Senilis. *See* CORNEA.

Argyria. *See* SKIN (Pigmentation of).

Arsenic. *See* TOXICOLOGY.

Arteries, Medical Affections of (including ATHEROMA and ARTERIO-SCLEROSIS). *See* VESSELS.

Arteries, Injuries of and Ligature of.

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GREEK ἀρτηρία = windpipe. These vessels were by the ancients supposed to contain air, as they are generally found empty after death. This error was exposed by Galen, who, by opening the arteries in a living animal, proved that they contained blood.

STRUCTURE.—The greater number of arteries are enclosed in a connective tissue sheath, to which the outer coat of the vessel is loosely connected by filamentous processes. So loose, however, is this connection that when an artery is completely divided transversely its cut extremities retract for some distance within the sheath. Apart from this sheath it is customary to describe an artery as having three coats, external, middle, and internal. *External coat* [Tunica adventitia]. This coat is composed chiefly of dense connective tissue with some admixture of yellow elastic fibres. The elastic tissue is most distinct in arteries of medium size and forms a more or less definite layer between the external and the middle coats of the vessel. *Middle coat* [Tunica media] consists of nonstriated muscular tissue arranged in a circular manner round the vessel. From this disposition it follows that when the middle coat is ruptured it tears in a circular direction. The thickness of the artery is due chiefly to the middle coat. It is a fact of importance that in the larger arteries there is a considerable admixture of connective and elastic tissue in the tunica media. Hence the contractibility of the arteries which is due to the muscularity of this coat is far more marked in the smaller vessels, and diminishes as the artery increases in size, becoming quite inconsiderable in the largest arteries. *Internal coat* [Tunica interna]. This coat is thin, transparent, colourless, and brittle, so that it readily breaks, particularly in the transverse direction. It consists of three layers:—

(1) A single endothelial layer, the cells of which have their long diameter in the direction of the vessel.

(2) The sub-endothelial, a delicate layer of branched corpuscles lying in an almost homogeneous matrix. This layer is most developed in the larger arteries, and is of great importance as being the seat of atheroma.

(3) An elastic membrane, arranged chiefly as a longitudinal network with numerous perforations, the fenestrated membrane of Henle. When an artery is ligated the ligature ruptures both the middle and internal coats. The outer coat, however, remains intact.

Vessels and nerves of arteries. The arterial coats receive small vessels, the vasa vasorum. These are distributed to the outer coat, but do not enter the middle or internal coats. Nerves are distributed to the coats of the vessels, but in health an artery is devoid of sensibility.

The INJURIES OF ARTERIES may be considered under the headings—1. Contusions. 2. Lacerations and Ruptures. 3. Punctured Wounds. 4. Incised Wounds. 5. Gunshot Wounds.

1. *Contusions of Arteries.*—A slight contusion of an artery may not give rise to any after-effects, or it may be attended by inflammatory changes in the vessel wall which lead to thickening of the coats of the artery and occlusion of its lumen. Should the contusion be sufficiently forcible to seriously injure the arterial wall the vessel may at the site of the injury become plugged by

an adherent thrombus, which may also eventually lead to obliteration of the artery.

2. (a) *Lacerated wounds* of arteries are apt to be attended with dangerous results, particularly secondary hæmorrhage should the wound become septic. There is as a rule but little primary hæmorrhage, for in these cases in which an artery is stretched beyond the limit of its elasticity and tears, the middle and internal coats snap across and curl up within the vessel, the middle coat contracting closes the orifice, and the overstretched external coat covers it. The danger in these cases is lest the injury sustained by the vessel walls just above the level of tearing across be sufficient to cause them to give way.

(b) *Rupture* of an artery may be partial, as, for example, when only the internal and middle coats of the vessel are severed whilst the external coat, though severely put on the stretch, remains intact. On the receipt of such an injury there is no primary hæmorrhage, but secondary hæmorrhage, the result of sloughing, or gangrene due to thrombosis of the vessel, are not uncommon results. If all three coats of an artery are completely torn across, the injury is that of a lacerated wound of the artery. From subcutaneous rupture of an artery a traumatic aneurysm may develop.

3. *Punctured wounds* of arteries are dangerous injuries, even though the wound be small in size. Any sharp-pointed instrument or splinter of glass, metal, etc., may cause a punctured wound of an artery. Hæmorrhage in such cases may not appear at once, or may be only trivial. Should, as not infrequently happens, the instrument which inflicted the injury introduce sepsis into the wound, septic arteritis, with ulceration and secondary hæmorrhage, will result. In punctured wounds of arteries the internal coagulum does not form, owing partly to the fact that retraction and contraction cannot occur as in an artery which is completely severed. The blood escaping from the hole in the artery becomes diffused through the tissues, as, owing to the small wound in the skin it cannot as a rule escape externally.

4. *Incised Wounds* of arteries are caused by sharp cutting instruments. Longitudinal wounds bleed but slightly. Transverse or oblique wounds bleed profusely when the artery is only incompletely divided, owing to the fact that contraction of the circular fibres of the middle coat causes the wound to gape wider than it would otherwise do. In such cases the artery should be cut completely across.

5. *Gunshot Wounds.*—A bullet may cause a contusion of an artery, may open into the lumen of the vessel, or may completely sever it.

Contused gunshot wounds of arteries may give rise to no primary hæmorrhage, but secondary hæmorrhage, especially in septic wounds, may be caused by sloughing of the arterial walls.

Perforation of an artery by a bullet generally causes profuse hæmorrhage. When the velocity of the bullet is sufficient to completely sever an artery, the division may resemble an incised wound and the hæmorrhage be formidable. Not infrequently simultaneous injury to an artery and its companion vein is caused by a gunshot wound. Gangrene or aneurysmal varix not infrequently follows such an injury. Foreign substances, *e.g.* pieces of clothing, are apt to be carried into the wound by the bullet and to set up infective inflammation therein.

The TREATMENT of injuries to arteries varies with the causes and symptoms of the lesion. In general any wound of an artery calls for ligature of the vessel above and below the point of injury, and complete division of the artery between the two ligatures.

In *contusions* complete rest and the maintenance of the warmth of the parts nourished by the artery are of prime importance in aiding in the establishment of the collateral circulation in cases in which thrombosis and obliteration of the vessel at the site of injury are apt to occur. The surgeon must ever be on his guard against the serious accident of secondary hæmorrhage.

In *lacerated wounds* and ruptures of arteries, even though the primary hæmorrhage is trivial or absent, the vessel should be ligated above and below the site of injury, owing to the risk of secondary hæmorrhage ensuing.

In *incised wounds* complete division of the wounded vessel after the application of a ligature above and below the incised wound should be the unvarying rule of practice.

In *gunshot injuries* the same treatment is called for. Owing to the danger of the supervention of sepsis the wound must be disinfected with scrupulous care. In gunshot wounds of an extremity the elastic bandage (Esmarch) is a valuable aid to the surgeon. Owing to the frequency of simultaneous injury to the artery and vein, gangrene is peculiarly liable to occur, and may rapidly prove fatal from septicæmia. Amputation may be necessary to avert death.

In cases in which the main artery and vein are injured, and in which the bone is shattered, amputation must be immediately performed.

In cases of gunshot wound in which the tissues in the neighbourhood of the injured artery become infiltrated with blood, and masses of coagulum press upon the vein, an incision should at once be made down to the injured artery, a ligature must be applied to it on either side of the lesion, and the blood-clot removed.

SIMULTANEOUS WOUND OF THE MAIN ARTERY AND VEIN of an EXTREMITY.—This, owing to the danger of gangrene supervening, is a most formidable accident. In such cases the wound in the artery must be secured by a ligature placed on either side of the bleeding point. Should the wound in the vein be but small, it should be sutured, or the margins of the orifice being caught up with artery forceps, they should be secured by lateral ligature in such a way as not to occlude the lumen of the vessel.

If the wound in the vein be a large transverse one, the vein must be doubly ligated. A long cut in the direction of the vein may generally be sutured with good result.

The limb should in all cases be wrapped in cotton wool and elevated.

WOUNDS OF SPECIAL ARTERIES

Common Carotid Artery.—A wound of this important vessel is commonly caused by a stab with homicidal, or more rarely suicidal, intent, a gunshot wound, or inadvertently by the surgeon during some operation in the neck. It is always a most dangerous, and not infrequently, unless surgical aid is at hand, an almost immediately fatal accident. The immediate danger to life depends upon the direction and extent of the wound in the vessel walls. A transverse cut almost completely severing the vessel is the most deadly, a small vertical incision parallel with the course of the vessel the least dangerous lesion, as in the latter compression may succeed in stanching the blood flow. In every case the artery must be securely ligated above and below the wound, and then completely severed. In suicidal cut-throat the artery, lying as it does deeply, is rarely injured. Secondary hæmorrhage from a wound of the carotid, occurring, as is usually the case, in a deep septic wound in the neck, is a very serious condition. If the ends of the vessel are in a sufficiently healthy state to allow of the application of ligatures, these should be placed on each extremity of the artery. In certain cases, however, all that can be done is to pack the wound tightly with antiseptic gauze, and if this fail, secure the vessel lower down and higher up.

External Carotid Artery.—In injuries of this artery, which are not infrequent, or in hæmorrhages from sloughing infected wounds or carcinomatous erosions of this vessel, *e.g.* in the case of secondary glandular growths, however difficult of execution the procedure may be, the proximal and distal ends of the divided or eroded vessel must be ligated. Ligation of the common carotid artery, a far easier and therefore more tempting operation, is in such cases quite unjustifiable, as it frequently fails permanently to arrest the hæmorrhage, owing to the very rich anastomosis of the branches of the external carotid, and it exposes the patient to the very serious risk of cerebral softening supervening on ligation of the common carotid artery.

Internal Carotid Artery.—Ligation of this artery is rarely called for, as wounds of this vessel are rare. Wound of the internal carotid during the performance of tonsillotomy, frequently described, is in practice unknown. In case of wound of the internal carotid artery a ligature should be applied, if possible, on each side of the lesion. If this cannot be accomplished, the common carotid artery may be tied with perhaps the external carotid in addition.

Internal Maxillary Artery.—This vessel, owing to its relation to the parotid gland and condyle of the mandible, defies successful ligation. The external carotid artery should be tied, and if hæmorrhage recur, pressure should be applied by gauze packing.

Lingual Artery.—This artery may be injured in bites of the tongue in epileptics, or secondary hæmorrhage may occur in sloughing wounds of the tongue after partial amputations of this organ. In every case, where possible, the vessel should be ligated at the bleeding point. Should this not be possible, however, from the rotten condition of the vessel walls, the artery must be tied at the seat of election in the neck.

Middle Meningeal Artery.—Hæmorrhage from a wound of this vessel is a not infrequent and formidable accident, and is usually observed in fractures of the cranium. The condition usually calls for trephining of the skull in simple fractures and ligature of the bleeding vessel. In compound fractures removal of the broken pieces of the skull may suffice without the further cutting of any more bone. It not seldom happens that, owing to its retraction into the bony canal in the parietal bone, or even into the foramen spinosum, this vessel defies ligation. In such cases the bleeding may be best arrested by plugging the bony canal with antiseptic wax, or failing this, with a wooden match made aseptic.

Axillary Artery.—Injury to this vessel is of not infrequent occurrence. Thus it may be wounded in dissecting malignant glands out of the axilla, in stabs, or ruptured in the reduction of dislocations of the upper end of the humerus of old standing. Here again double ligature will be required to control the alarming hæmorrhage, any part of the pectorals overlying the wound in the vessel being cut across, if necessary, to ensure free exposure of the bleeding point.

Subclavian Artery.—A wound partially severing this large vessel will produce a hæmorrhage so profuse that death may ensue in a few minutes. A punctured wound of small size will require the application of a ligature on either side of the wound.

Intercostal Artery.—Hæmorrhage from an intercostal artery is generally best treated by tightly plugging the wound with antiseptic gauze. Should it recur, it may require double ligature of the vessel after the resection of an inch of the rib if this is unavoidable.

Hæmorrhage from Wound of the Palm of the Hand.—This is of frequent occurrence, and in cases of secondary hæmorrhage in a septic sloughing wound is of really considerable danger. In primary hæmorrhage, if the superficial palmar arch be wounded, the best treatment is to apply a ligature on each side of the bleeding point if this can be found. Failing this, the wound having been carefully cleansed, an antiseptic compress may be employed. Should, however, the wound, as not infrequently happens, become infected, secondary hæmorrhage is in this part peculiarly prone to occur. In this case the unvarying rule of practice should be the application of a ligature to the brachial artery in the middle of the upper arm. Ligature of the radial and ulnar arteries is not nearly so likely to arrest the hæmorrhage, owing to the fact that the blood-flow in the two interosseous arteries and the arteria comes nervi mediani (which last is frequently of large size) will not be checked by ligature of the radial and ulnar arteries. Owing to the very free anastomosis around the elbow-joint there is no risk of the occurrence of gangrene after the ligation of the brachial artery.

Gluteal and Sciatic Arteries.—Stab wounds of these vessels in a large muscular buttock are, owing to the great depth at which they lie, always serious injuries. If possible the wound must be explored and two ligatures applied, one on either side of the bleeding point. Should, however, this last be in the pelvic portion of either vessel, the gravity of the injury is really formidable. The only course in such a case open to the surgeon is ligation of the parent trunk of the internal iliac artery.

Femoral Artery.—This vessel is of all the large arteries the one that is perhaps the most commonly injured. The bleeding point in case in which the hæmorrhage is not almost immediately fatal must be found, and a ligature applied to the vessel above and below the source of the hæmorrhage. In case of secondary hæmorrhage from a wound of the femoral artery, an attempt should be made to apply a ligature above and below those already tied on either side of the wound. Should the hæmorrhage recur, our choice lies between amputation at the level of the wound in the vessel and ligature of the external iliac artery. The former procedure, though one that the surgeon naturally would for many reasons wish to avoid, certainly has the lower mortality, and should therefore, as a rule, be adopted.

Popliteal Artery.—Owing to its depth this vessel generally escapes injury. It is, however, in some danger of being wounded in the removal of a sequestrum from the trigone of the femur. In the rare cases of wound of this vessel two ligatures, one on either side of the wound, should be applied. This is an operation of no little difficulty, owing partly to the depth of the artery from the surface, and also to the fact that this vessel is more closely adherent to its vein than any other artery in the body.

Anterior and Posterior Tibial Arteries.—Wounds of these vessels occur chiefly from the sharp fragments of bone in compound fractures of the leg. In such cases the wounded artery must be ligated above and below the bleeding point.

LIGATION OF ARTERIES IN CONTINUITY

The ligation of an artery in its continuity is an operation which any practitioner may be called upon to perform at a moment's notice under the most unfavourable conditions, without professional assistance and with but few instruments at hand, and yet, in not a few instances, the life of the patient will depend upon the promptness and judgment of the operator. There are few things more easy to forget than the relations of the main arteries, and yet every medical man, having regard to the fact that he may suddenly be called upon to ligature one of these vessels, should consider it his duty to keep in remembrance the anatomy of at least the more important arteries. It has been wisely said that there is no such thing as surgical anatomy, that all anatomy is surgical. To no section of anatomy does this apply with greater force than to that of the main arteries. It need hardly be impressed upon the reader that, owing to the tremendous risks of secondary hæmorrhage in septic arteritis, every operation for the ligation of an artery in its continuity must be conducted with the most scrupulous antiseptic precautions.

Method of Ligation.—The incision is made in the direction of the course of the vessel, and may vary, according to the size of the artery and the depth at which it lies from the skin surface, from 1 to 6 inches in length. In the upper and lower limbs the line of incision corresponds with the long axis of the limb. The incision having been carried through the skin and subcutaneous tissue, the deep fascia is divided in the whole length of the wound and the sheath of the artery is exposed, muscles, veins, and nerves which may overlie the artery being drawn aside with blunt retractors. The sheath of the vessel must now be opened with care on the side opposite to that on which the vein corresponding to the artery lies. A small piece of the sheath is held up by forceps and an opening is made into it; the sheath is then slit longitudinally for $\frac{1}{3}$ inch. It is necessary that the sheath be freely opened, for although it is important to detach it as little as possible from the artery, owing to the fact that the vasa vasorum run in it, free linear division of the sheath will entail no risk, and will much facilitate the passing of the aneurysm needle around the artery. The sheath having been opened, it is well to seize each margin of the orifice in it with fine forceps in order to secure this opening, as it may easily be lost sight of. The artery must be identified by its position and anatomical relations, as in certain cases all other means of identification may prove fallacious. It might be thought that pulsation would enable the operator to identify unhesitatingly the artery, and no doubt this is generally the case; but after a profuse hæmorrhage pulsation is not infrequently absent, and, moreover, a vein may appear to pulsate owing to the impulse being transmitted from the artery. The artery having been identified, the operator isolates the circumference of the vessel for ¼ of an inch, in case only one ligature is to be applied, or for ½ inch if it is considered desirable to employ double ligation with division of the artery between the two ligatures. The aneurysm needle, threaded, should then be gently insinuated between the sheath and the artery from that side of the vessel nearest to which the vein is situated. As soon as the blunt end of needle bearing the ligature is visible on the other side of the artery, the thread is seized with forceps and the needle withdrawn, care being taken not to lift the artery out of its sheath. If thought desirable, a second ligature may now be passed in a similar way about ⅓ or ½ inch below the first thread. The ligatures are then tightened and secured by a reef knot, or better, by a surgical knot, *i.e.* one in which in making the first half of the knot a double twist is given instead of only a single twist as in the reef or sailor's knot. In case two ligatures are applied, they should be placed at a distance of ⅓ to ½ inch apart, and the artery should be divided between them. By the employment of double ligation with division of the artery between the ligatures, the retraction of the divided ends takes off all longitudinal strain from the artery, but it is doubtful if the method possesses advantages sufficient to recommend its employment in ordinary cases.

Points to which special attention must be directed in the ligature of an artery are:—

1. Each successive incision from the skin surface to the sheath of the vessel must be made of the same length.

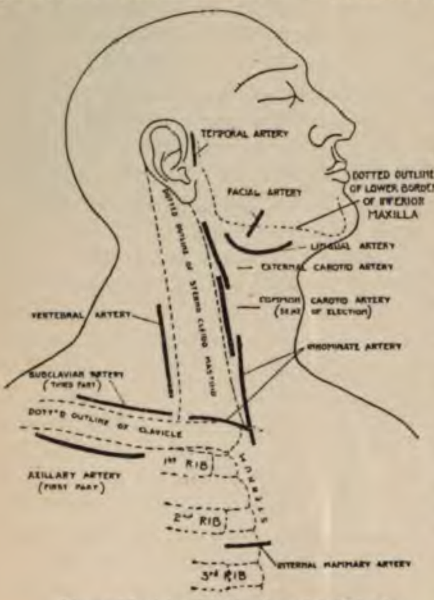
2. The sheath must be freely opened and separated from the vessel in the whole of its circumference. This separation may, I think, be most safely accomplished by gently insinuating the blunt point of the aneurysm needle between the sheath (held tense by forceps) and the wall of the vessel. This manœuvre must be carried out gradually with extreme gentleness.

3. Before the ligature is tied, the operator must make certain that when the vessel is ligated the pulsation will be absent upon the distal side of the vessel (*e.g.* in an aneurysm). This may be ascertained by pressure of the index finger upon the vessel, whilst at the same time the extremities of the thread are made tense. Thus the artery is compressed between the forefinger in front and the ligature behind.

4. A perfectly sterilised ligature must be employed, and the operation conducted with every precaution against wound infection. The three materials generally employed for ligature are silk, chromic catgut, and kangaroo tendon. Silk has the great advantage that in an emergency it can be rapidly sterilised by boiling.

Arteries, Ligature of Special

Innominate Artery.—Ligation of this large vessel is, as will be readily understood, a very formidable undertaking. The mortality of the operation has, even taking into consideration recent unpublished cases, been something like 90 per cent. The most frequent cause of death has been secondary hæmorrhage. The innominate artery arises from the upper aspect of the aortic arch, and terminates after ascending obliquely to the right opposite the upper margin of the right sterno-clavicular joint, at which level it bifurcates into the right common carotid and right subclavian arteries. Its average length is 1½ inches.


TEMPORAL ARTERY
FACIAL ARTERY
DOTTED OUTLINE OF LOWER BORDER OF INFERIOR MAXILLA
DOTTED OUTLINE OF STERNO CLEIDO MASTOID
LINGUAL ARTERY
EXTERNAL CAROTID ARTERY
COMMON CAROTID ARTERY (SEAT OF ELECTION)
VERTEBRAL ARTERY
INNOMINATE ARTERY
SUBCLAVIAN ARTERY (THIRD PART)
DOTTED OUTLINE OF CLAVICLE
1st RIB
AXILLARY ARTERY (FIRST PART)
2nd RIB
INTERNAL MAMMARY ARTERY
3rd RIB

Lines of Incision for Ligation of Arteries in Head and Neck.

Operation.—The incision is made along the anterior margin of the right sterno-cleido-mastoid muscle in its lower two-fifths. This should extend to just below the supra-sternal notch, and from the lower end of the incision a cut is to be made outwards for 1½ to 2 inches along the upper margin of the clavicle. The superficial layer of cervical fascia and sternal head of the sterno-cleido-mastoid muscle having been divided, the anterior jugular vein is drawn aside and the sterno-hyoid and sterno-thyroid muscle retracted inwards, or better, cut across. The second layer of cervical fascia (pericardial) is now carefully incised, and the right inferior thyroid vein divided after double ligation. The forefinger of the surgeon now can readily feel the innominate artery at its bifurcation. The pleura lies behind and to the right of the vessel, the left innominate vein crosses in front of the innominate artery at its lower end, and the right innominate vein lies on its right side. The right pneumogastric nerve with its recurrent laryngeal branch and the right phrenic nerve lie to the right (outer) side of the vessel, but may require to be drawn farther outwards whilst the ligature is being placed. The aneurysm needle, threaded with a thick sterilised silk ligature, should be passed from without inward with a tendency upwards in order to avoid the pleura, which is the structure most liable to injury in this operation. In the place of a silk ligature many surgeons recommend a kangaroo-tail tendon.

Ligature of the Common Carotid Artery.—The two common carotid arteries have almost identical courses in the neck. The difference in their origin scarcely affects their ligation, as this is always carried out in the cervical part of the vessel (in the case of the left artery). The course of each vessel, from a surgical point of view, may be said to be from the upper margin of the sterno-clavicular articulation to the upper border of the thyroid cartilage, in a line from the sterno-clavicular joint to a point midway between the angle of the jaw and the mastoid process. The artery is crossed at the level of the lower margin of the cricoid cartilage by the omo-hyoid muscle, and the seat of election for ligation of the vessel is just above the omo-hyoid, *i.e.* about the level of the cricoid cartilage. The cervical portion of the artery is about 3¾ inches in length, and can be felt pulsating in its whole course on either side of the larynx and trachea.

The more important relations of the vessel are :—Externally, the internal jugular vein ; internally, larynx, pharynx, trachea, and recurrent laryngeal nerve ; posteriorly, vagus nerve and sympathetic.

Operation (at seat of election).—An incision 3 inches in length is made along the anterior border of the sterno-mastoid muscle, the centre corresponding with the cricoid cartilage. The skin, superficial fascia, fibres of platysma, and the deep fascia are divided, and the anterior margin of the sterno-cleido-mastoid is now clearly defined and retracted outwards, when the carotid artery will be found encased in its sheath in the angle formed between that muscle and the omo-hyoid which crosses the vessel. The descendens hypoglossi nerve is generally seen running downwards upon the sheath, and it is drawn aside and the sheath carefully opened on its antero-internal aspect. The internal jugular vein generally somewhat overlies the artery, and this is particularly the case lower down in the neck, hence the necessity of opening the sheath on its antero-internal aspect. Great care is requisite in separating the artery from the sheath and in avoiding injury to the pneumogastric nerve, which lies behind the vessel, and to the internal jugular vein on its outer aspect. The aneurysm needle should be passed from without inwards.

It may happen that the surgeon is forced to ligature the artery low down in the neck. In this case a 4-inch incision is made in the line of the vessel lower in the neck. The anterior margin of the sterno-mastoid having been defined, it is drawn outwards, and the sterno-hyoid and sterno-thyroid muscles are retracted inwards or partially divided. The omo-hyoid is now seen lying external to the former muscles; it is drawn outwards. The carotid artery is now felt for in the angle between the omo-hyoid and sterno-thyroid. The sheath having been opened still more on the inner side owing to the overlapping of the vessel (especially on the left side of the neck) by the internal jugular vein, the aneurysm needle is passed from without inwards, avoiding, in addition to the structures mentioned above, the recurrent laryngeal nerve.

Collateral circulation.—After ligation of the common carotid artery the circulation is carried on by the anastomosis of the following vessels:—

1. Internal carotid with internal carotid of the other side through the circle of Willis.
2. Vertebral artery with vertebral artery of other side through circle of Willis.
3. Superior thyroid artery (from external carotid) with inferior thyroid artery (from thyroid axis of subclavian) of the *same* side.
4. Arteria princeps cervicis (from occipital) with deep cervical artery (from superior intercostal) of the same side.
5. Branches of the external carotid, especially facial and superior, thyroid with branches of external carotid of the other side.

The chief *complications* which are apt to follow ligature of the common carotid artery are cerebral symptoms referable to the interference with the blood-supply to the brain. These are very frequent, and vary from giddiness and twitching to hemiplegia and even death. So serious are the cerebral symptoms that it must be laid down, as a rule in surgery, that ligature of the common carotid artery is not justifiable in any case in which ligature of the external carotid would with equal certainty arrest the hæmorrhage or cure the condition requiring ligation of one of these arteries. Apart from septic complications which are avoidable, a low form of pneumonia, possibly due to interference with the blood-supply to the respiratory centre in the medulla oblongata, has been noticed not infrequently.

Ligature of the External Carotid Artery.—This vessel, one of the two divisions of the common carotid artery, reaches from the upper border of the thyroid cartilage upwards to the level of the neck of the condyle of the inferior maxilla. Its length is 2¾ inches. This vessel, about the same size as the internal carotid artery, lies at first in front of the latter; it, however, soon comes to lie superficial to it. In its upper part it lies embedded in the parotid gland. The lowest inch of the artery, *i.e.* below where it is crossed by the digastric and stylo-hyoid muscles, and the hypoglossal nerve, is that part of the vessel to which alone a ligature is applied.

Operation.—The incision lies along the anterior border of the sterno-cleido-mastoid, from the level of the angle of the jaw to the lower margin of the thyroid cartilage. The skin, fascia, and platysma muscle having been divided without injury to any of the numerous veins (facial, lingual, and superior thyroid), the artery is readily seen, crossed by the digastric and stylo hyoid muscles, and the hypoglossal nerve. It is possible to mistake the internal carotid for the vessel, an error that can easily be avoided by finding a branch arising from the external carotid (the internal carotid gives off no branch in the neck). The artery is best tied ¾ inch above the bifurcation of the parent trunk, care being taken to avoid including in the ligature the internal jugular vein, which lies to its outer side, but may overlap the artery. The aneurysm needle is passed from without inward.

Ligature of Internal Carotid Artery.—This is an operation very rarely called for. The incision required is identical with that described for the external carotid. The artery lies at first behind the external carotid, and in securing it the aneurysm needle requires to be passed from without, inward, *i.e.* away from the internal jugular vein; care must also be taken to avoid including in the ligature the vagus nerve.

Ligature of the Lingual Artery.—This is an operation which the surgeon is frequently called upon to perform, generally as a preliminary to removal of the tongue. It may also be required for profuse hæmorrhage from the tongue in advanced lingual epithelioma. The artery arises from the external carotid, opposite the great cornu of the hyoid bone. In its course to the tongue it lies under cover of the hyoglossus muscle, and along the superior margin of the great cornu of the hyoid. The vessel near its origin is crossed by the hypoglossal nerve, which then runs forwards at a higher level than the artery and superficial to the hyoglossus muscle.

Operation.—The chin having been drawn up and turned to the opposite side, and the great cornu of the hyoid bone having been identified, a curved incision is to be made from just below the angle of the jaw to the hyoid bone, and thence upward to a point ½ inch below, and external to the symphysis of the lower jaw. The skin, platysma, and fascia having been divided, the lower part of the submaxillary gland is displayed and drawn upwards, when a triangle is exposed, whose base, which is uppermost, is the hypoglossal nerve, and whose sides are the anterior and posterior bellies of the digastric which meet below at the apex. The floor of the triangle is the hyoglossus muscle. If now the fibres of the hyoglossus be divided transversely below the hypoglossal nerve and above the great cornu of the hyoid, the lingual artery will be found lying on the middle constrictor muscle. Provided that the hypoglossal nerve is avoided, it is immaterial whether the aneurysm needle be passed from above downward, or from below upward.

Ligature of the Superior Thyroid Artery.—This, the first branch of the external carotid artery, arises from it just above the bifurcation of the common carotid artery. It is frequently divided in suicidal cut-throat, when of course both ends of the cut vessel must be ligated. In thyroidectomy it is frequently ligated prior to removal of the thyroid gland.

Operation.—A three-inch incision is made along the anterior border of the sterno-cleido-mastoid muscle with its mid point just above the upper margin of the thyroid cartilage. The skin, platysma, and fascia being now divided, the sterno-cleido-mastoid is retracted, when the carotid sheath is at once seen, and the first branch arising from the external carotid will be found to be the superior thyroid artery entering the thyroid gland. The superior thyroid, lingual, and facial veins are, as in all operations in this region, very liable to be injured. The aneurysm needle may be passed from above downward.

Ligature of the Facial Artery.—This, the third branch of the external carotid artery, may be exposed in the neck by an incision similar to that mentioned for the superior thyroid, but rather higher up. It must be borne in mind that frequently the facial and lingual arteries arise by a common trunk from the external carotid. The artery is, however, most readily ligated where it crosses over the lower jaw in front of the masseter muscle.

Operation.—A vertical incision just in front of the masseter, dividing skin, fascia, and platysma, serves to expose the artery emerging from under cover of the submaxillary gland, and crossing the inferior border of the lower jaw. As the facial vein lies on its outer side the needle is passed from without inwards.

Ligature of the Superficial Temporal Artery.—This vessel, one of the two terminal branches of the external carotid artery, continues upwards in the course of the parent trunk, and crosses the zygoma between the condyle of the lower jaw and the tragus of the ear.

Operation.—A vertical incision one inch in length and ½ inch in front of the tragus, will expose the artery. The tissues here are very dense and firm. The pulsation of the artery can be readily felt on the zygomatic arch. The needle is to be passed from behind forward in order to avoid the accompanying vein and the auriculo-temporal nerve, which lie between the artery and the tragus.

Ligature of the Subclavian Artery.—The subclavian artery on the right side arises from the bifurcation of the imnominate artery; on the left side it springs from the arch of the aorta. In the lowest part of the neck on both sides it arches outwards above the pleura and behind the scalenus anticus. The first part of the vessel reaches from its origin to the internal border of the scalenus anticus muscle; the second part lies under cover of the same muscle; and the third part reaches from the external border of the scalenus anticus to the external margin of the first rib. The artery is tied almost without exception in the third part of its course. Ligation of the first part of the artery is among the most fatal operations in surgery, and from the records of past cases is manifestly unjustifiable. Ligation of the second part of the artery may be performed by extending inwards the incision made for ligature of the third part, by incising transversely, or even completely dividing the anterior scalenus muscle. *Ligation of the third part of the artery* is the operation almost invariably performed.

The more important relations of the third part of the subclavian artery are:—

In front: subclavian vein on a lower level, the external jugular and its tributary veins.

Above: cords of brachial plexus.

Below: the first rib.

Behind: the scalenus medius muscle, and, generally, the lowest cord of the brachial plexus.

An important landmark is the outer margin of the sterno-cleido-mastoid, which almost exactly corresponds with the outer margin of the scalenus anticus muscle. Just external to this line, the artery may be securely compressed against the first rib.

Operation.—To ligature the third part of the subclavian artery, the shoulder is depressed, and the head turned to the opposite side. The skin is now drawn down over the clavicle and an incision is made along that bone from the outer border of the sterno-cleido-mastoid, to the anterior border of the trapezius muscle. In making the incision thus, the external jugular vein is drawn under cover of the clavicle and avoids the risk of injury. When the skin resumes its former position the incision will lie well above the clavicle and directly over the artery. The deep fascia is now divided, particular care being taken to avoid all veins, especially the external jugular vein. If any vein cannot, however, be drawn aside, it must be divided between two ligatures. This is important, as in case of injury to the external jugular, air may be sucked into it during inspiration. Guided by the finger, and using a dissector or dissecting forceps, the loose cellular and fatty tissue is now opened up, and the omo-hyoid muscle exposed and drawn upwards. The outer margin of the anterior scalene muscle can now readily be defined, and if traced downwards will lead the operator to the scalene tubercle on the first rib, behind which lies the artery which can be readily felt pulsating. The artery is now carefully isolated, and the aneurysm needle passed from above downwards and from behind forwards. It will be observed that this is not from the vein, according to the general rule, but as a matter of fact the great risk in passing the thread around the artery is not injuring the vein which lies in front and at a lower level, but of including in the ligature the lowest cord of the brachial plexus. There is some danger also of puncturing the pleura unless extreme gentleness be employed.

Should it be necessary, owing, for example, to the wall of the vessel being unhealthy in its third part, to ligature the artery in its second stage, the scalenus anticus may be divided in its outer two-thirds and the second part of the artery ligated. Special care is necessary to avoid injury to the phrenic nerve which lies on the anterior aspect of the muscle.

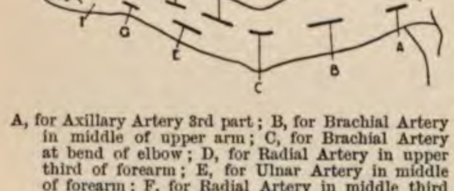
Collateral circulation after ligature of third part of subclavian artery is chiefly carried on by anastomoses around the scapula between the supra-scapular, posterior-scapular, and sub-scapular arteries, and also by twigs from the internal mammary, superior intercostal, and aortic intercostal arteries anastomosing with the long thoracic artery and the above-mentioned scapular arteries.

Ligature of the Vertebral Artery.—An incision along the hinder border of the sterno-cleido-mastoid, commencing one inch above the clavicle, and passing upwards for four inches, is made, avoiding injury to the external jugular vein. The sterno-cleido-mastoid is drawn forwards, when the scalenus anticus muscle will be defined with the phrenic nerve lying on its anterior aspect. The interval between this muscle and the longus colli is now defined, and the anterior tubercle of the tranverse process of the sixth cervical vertebra, the deep guide to the vessel, is readily felt. Just below this bony point in the inter-muscular gap, the vertebral vein is found lying superficial to the artery. The vein is retracted a little outwards to allow of the needle being passed from without inwards.

Ligature of Inferior Thyroid Artery.—An incision is required three inches in length, running upwards along the anterior margin of the sterno-cleido-mastoid muscle, and commencing half an inch above the clavicle. The sterno-cleido-mastoid muscle and the carotid sheath are now drawn outwards. The operator next makes out the anterior tubercle of the transverse process of the sixth cervical vertebra, and the artery is found coursing inwards just below this bony point. Great care must be exercised in isolating the vessel, as the middle cervical (thyroid) ganglion of the sympathetic frequently lies upon the artery.

Ligature of Internal Mammary Artery.—This vessel may be ligatured by means of a transverse incision one inch and a half in length, the centre of the incision being placed half an inch outside the margin of the sternum. In the upper part of its course the vessel lies directly on the pleura, in the lower part the triangularis sterni intervenes between it and this membrane. In order, therefore, to reach the artery, the superjacent fibres of the pectoralis major, the anterior intercostal membrane, and the internal intercostal muscle must be divided. Both ends of the vessel, if completely divided, must be found and secured with ligatures.

Ligature of the Axillary Artery is seldom called for except for stab wound. The vessel commences at the outer margin of the first rib, and terminates by becoming the brachial artery at the lower border of the teres major muscle, *i.e.* at the lower margin of the posterior axillary fold. When the arm is abducted so as to lie at a right angle with the trunk, a line drawn from the centre of the clavicle to the inner margin of the coraco-brachialis muscle (a prominence always to be made out in this position of the limb) will mark the course of the artery. The vessel may be tied in either the first, second, or third part of its course, *i.e.* internal to, beneath, or beyond the pectoralis minor muscle. Ligature of the first and third parts only call for separate description.



A, for Axillary Artery 3rd part; B, for Brachial Artery in middle of upper arm; C, for Brachial Artery at bend of elbow; D, for Radial Artery in upper third of forearm; E, for Ulnar Artery in middle of forearm; F, for Radial Artery in middle third of forearm; G, for Ulnar Artery just above wrist; H, for Radial Artery just above wrist; I, for Superficial Palmar Arch.

Ligature of the First Part of the Axillary Artery.—A transverse, slightly curved incision with convexity downwards is made, corresponding in length with the middle two-fourths of the clavicle, and placed half an inch below the bone. The interval between the clavicular part of the pectoralis major muscle and the deltoid is now sought and opened, and the clavicular fibres of the great pectoral are divided close to the bone. The costo-coracoid membrane is now exposed and is divided transversely half an inch below the clavicle, and when by blunt dissection the areolar tissue has been cleared to either side, the artery is found deeply placed, with the corresponding vein lying partly on its inner side and partly superficial to it, whilst the cords of the brachial plexus are placed on its outer side. The vein being drawn inwards, the aneurysm needle is passed from below and within upwards and outwards.

Ligature of the Third Part of the Axillary Artery.—The arm being abducted, an incision three inches in length is made in the line of junction of the anterior third with the middle third of the axillary space between the anterior and posterior folds. This corresponds below to the inner margin of the prominence caused by the coraco-brachialis muscle. This muscle, being exposed by division of skin and fascia, is drawn outwards, when the artery will be found with the median and musculo-cutaneous nerves on its outer side, the inner head of the median nerve superficial to it, and the ulnar and internal cutaneous nerves and the axillary vein on its inner side. The median nerve having been drawn outwards, the needle is to be passed from within outwards, *i.e.* from the vein.

Collateral Circulation.—If the first part of the artery is ligated above the origin of the acromio-thoracic artery, this is identical with that for the ligature of the third part of the subclavian. If the third part of the artery is ligated below the circumflex arteries, the circulation in the upper limb will be carried on by anastomosis of the subscapular and circumflex arteries with the superior profunda.

Ligature of the Brachial Artery.—This vessel continues the course of the axillary artery, and reaches from the lower margin of the teres major to a point opposite the neck of the radius, where it bifurcates into the radial and ulnar arteries. It is remarkably superficial in its whole course, except just near its bifurcation. The artery is accompanied by two venæ comites, and is crossed from without inwards by the median nerve about the middle of the upper arm. The artery may require ligature for wound of the vessel, for palmar hæmorrhage, or for aneurysm in the region of the elbow.

Ligature of the Brachial Artery in the Middle of the Arm.—The arm being held at a right angle to the trunk and the forearm supinated, an incision three inches in length is made through skin and superficial fascia along the inner margin of the biceps muscle. The basilic vein, thus exposed, is drawn aside, and the deep fascia cautiously divided. The artery is now found with the median nerve crossing it. The nerve is to be drawn aside (generally outwards), the venæ comites are separated from the artery, which is then ligated, the needle being passed from the nerve. It must be borne in mind that the nerve may cross behind the artery, and that the vessel itself may have divided high up in the arm. There will in this case be two arteries to ligature in place of one. The inner margin of the biceps must always be most carefully defined, and the arm must not rest on the table, otherwise the triceps will be pushed up so as to partly cover the artery.

Ligature of the Brachial Artery at the Bend of the Elbow.—An incision, oblique, from within outwards and downwards, and two inches in length, is made parallel to the inner margin of the biceps tendon. The skin, fascia, and bicipital aponeurosis having been divided, the artery will be found having the biceps tendon on its outer side, and the median nerve some little distance away on its inner side. The needle is passed from within outwards.

Collateral Circulation.—If, as commonly happens, the ligature is applied to the artery below the origin of the inferior profunda branch, the superior and inferior profunda arteries from above anastomose with the radial recurrent, the anterior and posterior ulnar recurrent, the interosseus recurrent, and the anastomotica magna arteries.

Ligature of Arteries in the Forearm.—Spontaneous aneurysm being below the brachial so rare that its existence is almost doubtful, and traumatic aneurysm being very rare, it follows that ligature of the radial and ulnar arteries will only, save under very exceptional circumstances, be required for hæmorrhage following injury to these vessels. In all such cases the wounded vessel must be secured by ligature above and below the bleeding point.

LIGATURE OF THE RADIAL ARTERY.—A line drawn from just below the centre of the bend of the elbow to a point half an inch internal to the styloid process of the radius will indicate with sufficient accuracy the *course of the radial artery in the forearm.*

Ligature in Upper Part of Forearm.—An incision two to two and a half inches long is made in the line of the vessel, and the intermuscular space between the supinator radii longus and the pronator radii teres is widened by retracting outwards the inner border of the former muscle, under which lie the radial artery, and its venæ comites. The radial nerve is in close relation with the artery on its outer side only in the middle third of the forearm.

Ligature in the Lower Part of the Forearm.—Just above the wrist is the seat of election for ligature of this vessel. Here the artery with its two venæ comites lies very superficial, covered only by skin and a thin fascia, between the tendons of the supinator radii longus and flexor carpi radialis. An incision one and a half inches in length readily exposes the vessel. The radial nerve has at this place left the artery to pass to the dorsum of the wrist.

LIGATURE ON THE DORSUM OF THE WRIST.—The radial artery is readily ligated in the so-called "tabatière anatomique," where it lies between the tendons of the extensor primi internodii and secundi internodii pollicis in its course towards the proximal end of the first interosseous space.

An incision one and a half inches in length is made parallel with the former tendon and to its ulnar side. The radial vein is avoided, and the deep fascia being incised, the artery is found and ligated. Great care must be taken to avoid wounding the tendon sheaths, otherwise adhesions may form between the tendons and neighbouring structures.

LIGATURE OF THE ULNAR ARTERY.—The course of the vessel corresponds to a line drawn from just below the centre of the bend of the elbow to the radial side of the pisiform bone. In the upper third of its course in the forearm it is directed obliquely downwards and inwards, and placed so deeply that its ligature here cannot be required. The vessel may be ligatured about the middle of the forearm or above the wrist. In the former position the vessel is overlapped by the flexor carpi ulnaris, which lies on its inner side. Just above the wrist it is superficially placed between the tendon of the flexor carpi ulnaris on its ulnar side, and the tendons of the flexor sublimis digitorum on its radial aspect. The ulnar nerve in the lower two-thirds of the forearm lies closely applied to the inner side of the artery.

Ligature in the Middle of the Forearm.—An incision three inches in length is made in the line of the vessel. The deep fascia being divided, the intermuscular space (which is generally by no means easy to discover) between the flexor carpi ulnaris and flexor sublimis digitorum muscles is opened, when the artery with its venæ comites is found lying on the flexor profundus digitorum, with the ulnar nerve lying on its inner side. The nerve being drawn inwards, the needle is passed from within outwards.

Ligature just above the Wrist.—An incision extending upwards for one and a half inches is made in the line of the vessel commencing just above the wrist. The deep fascia is incised, the artery is found lying to the radial side of the flexor carpi ulnaris tendon, with the ulnar nerve to its ulnar side. The venæ comites being detached from the artery, the aneurysm needle is passed from within outwards.

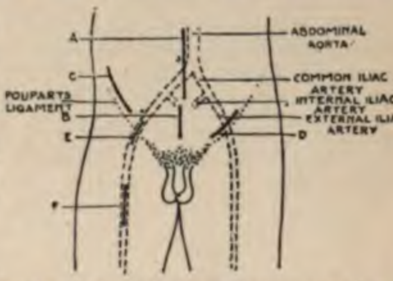
LIGATURE OF THE SUPERFICIAL PALMAR ARCH.—As mentioned when treating of palmar hæmorrhage, we have two different conditions to deal with: (1) the arrest of primary hæmorrhage; (2) the treatment of secondary hæmorrhage in an infected sloughing wound.

In the former case, careful cleansing of the wound with the application of an antiseptic compress will generally procure the cessation of the bleeding. In certain cases, however, in which the artery has been only partially divided, hæmorrhage cannot be controlled by pressure.

Operation.—The vessel must be exposed and divided between two ligatures applied on either side of the wound in the artery. An Esmarch's elastic bandage should always be made use of to allow a good examination of the part. The arch lies immediately under cover of the palmar fascia, and in the middle third of the palm lies at the same level as the upper end of the cleft between the thumb and index finger. The mid point of the arch is practically the centre of the palm. The nerves and tendons lie beneath the arterial arch. The superficial wound is to be enlarged, the wound in the artery found, and in every case both ends of the divided or partially divided vessel securely ligated.

In secondary hæmorrhage from an infected wound of the palmar arch, ligature of the brachial artery in the middle of the arm is undoubtedly the wisest treatment. So free is the anastomosis around the elbow that no fear of gangrene of the hand need be entertained.

Ligature of Abdominal Aorta.—This operation has proved fatal in every case in which it has been undertaken, though in one case (Monteiros's) the patient lived ten days. The abdominal aorta commences in front of the middle of the last dorsal vertebra, and terminates by dividing into the two common iliac arteries on the left side of the fourth lumbar vertebra, *i.e.* just below and to the left of the umbilicus. On its right side in its lower two-thirds lies the vena cava inferior.



A, for Abdominal Aorta (transperitoneal); B, for Iliac arteries (transperitoneal); C, for External Iliac artery (retro-peritoneal); D, for External Iliac artery (retro-peritoneal); E, for Common Femoral artery; F, for Superficial Femoral artery (seat of election).

Two methods may be mentioned: (1) the old retro-peritoneal; (2) the newer transperitoneal operation. 1. *The retro-peritoneal* method. An incision is made reaching from the tip of the tenth rib of the left side downwards to the crest of the ilium on the same side. This incision divides the flat muscles of the abdomen until the fascia transversalis is seen. This is then incised vertically, care being taken to avoid opening the parietal peritoneum, which is to be carefully stripped off the ilio-psoas muscle by the hand passing downwards and backwards until the aorta is felt pulsating in front of the spine. It is excessively difficult to isolate the artery so as to pass a ligature around it. This method is difficult, uncertain, and dangerous, as it involves working in the dark. It would never be employed by the surgeon of to-day.

2. *Transperitoneal Method.*—A six-inch vertical incision is made with its centre just to the left of the umbilicus. The abdominal parietes with the peritoneum having been divided and the small intestine pushed to the right side, the aorta comes into view clothed by the parietal peritoneum which covers the posterior wall of the abdomen. This membrane is now carefully divided and the aorta cleaned. The aneurysm needle should now be passed from right to left, *i.e.* from the vein.

Ligature of the Common Iliac Artery.—Each vessel is only about two inches in length. The common iliac arteries commence by the bifurcation of the abdominal aorta on the left side of the body of the fourth lumbar vertebra, and terminate on the level of the upper margin of the sacrum by dividing into the external and internal iliac arteries. The common iliac artery lies under cover of the peritoneum, and is crossed just at its bifurcation by the ureter. The right common iliac vein near the bifurcation is placed under cover of the artery, but higher up lies on its right side. The left common iliac vein comes into relation with both common iliac arteries; below, the vein lies to the right side of its own artery, and higher up, in order to reach the vena cava inferior, it passes from left to right under cover of the right common iliac artery. These complicated venous relations indicate the absolute necessity of the transperitoneal methods in which alone the vessels can be readily seen. The course of the common and external iliac arteries may be indicated with sufficient accuracy by a line drawn from a point half an inch below and half an inch to left of umbilicus to a point midway between the anterior superior iliac spine and the symphysis pubis. The uppermost two inches of this line correspond to the common iliac, the remainder to the external iliac artery.

The Transperitoneal Method.—An incision four inches in length (in a thin patient one inch less) is made in the mid line of the abdomen, or better, in the corresponding semilunar line (outer margin of rectus abdominis muscle) commencing one inch above Poupart's ligament and directed upwards. The parietes and peritoneum having been divided, the small intestines are pushed aside, and the pulsations of the artery being readily felt, the peritoneum covering the vessel is incised and the artery cleaned. The aneurysm needle is to be passed from the vein. If the vessel be ligated just above its bifurcation, care must be exercised lest it be included in the ligature or otherwise injured.

Retro-peritoneal Method.—In this operation the peritoneum is stripped up so as to ligate the artery without incising the peritoneal membrane. An incision about six inches in length commences one inch above the mid point of Poupart's ligament, and is curved upwards and outwards so as to lie one inch internal to the anterior superior iliac spine and to terminate one or two inches above this point. The parietes are divided down to the parietal peritoneum. This is then to be detached in an upward and forward direction from the iliac fossa. The external iliac artery is now readily demonstrated, and by following this vessel upwards the common iliac trunk is reached. The areolar tissue surrounding the artery is carefully separated and the aneurysm needle passed from the vein, *i.e.* on the left side from within outwards, on the right side from without inwards.

The ureter is rarely in the way, as it almost invariably adheres to the peritoneum and is raised with it. This operation labours under the disadvantage of giving an insufficient view of the artery and vein, and is distinctly more dangerous and difficult than the transperitoneal ligature, which is always to be preferred.

LIGATURE OF EXTERNAL ILIAC ARTERY.—This is the largest artery which is commonly ligated by the surgeon, and, considering the magnitude of the vessel the results of ligature of the external iliac artery are remarkably good. The vessel reaches from the bifurcation of the common iliac artery and extends outwards to Poupart's ligament. The course of the common and external iliac arteries is sufficiently indicated by a line drawn from a point half inch below and to left of umbilicus to a point midway between the anterior superior iliac spine and the symphysis pubis. The upper two inches of the line represents the common iliac artery, the remaining portion the external iliac artery. The artery is in its whole course retro-peritoneal, the corresponding vein lies upon the inner side of the artery except that at the upper end of the right artery the vein lies behind its artery, and the genito-crural nerve on its outer side. Just above Poupart's ligament the genital branch of the last-named nerve, the deep circumflex iliac vein, and the spermatic artery cross the vessel. The chief indications for ligature of this artery are aneurysm in the groin and secondary hæmorrhage from the femoral artery.

Operation.—An incision, four inches in length, commences half an inch above and just external to the centre of Poupart's ligament, and extends outwards and upwards to a point one inch internal to, and one inch above the level of the anterior superior spine of the ilium. The skin and fascial aponeurosis of the external oblique, internal oblique, and transversalis muscles are divided in the whole length of the wound. The fascia transversalis now comes into view, and is most carefully incised without injury to the peritoneum. The latter membrane is now separated by means of the fingers of the operator from the fascia iliaca and drawn upwards and inwards. The fingers can now feel the pulsation of the vessel which lies still covered by the fascia along the inner margin of the psoas muscle. The surgeon now opens the fascia over the artery one to one and a half inches above Poupart's ligament, isolates the vessel from its sheath, and passes the aneurysm needle from within outwards to avoid injury to the vein.

The artery may also be tied by the *transperitoneal method* through an incision in the lower part of the semilunar line. See ligature of common iliac artery. This method enables the surgeon more certainly to avoid injury to the vein. In the extra-peritoneal operation the peritoneum is not infrequently opened in error.

The artery may also be ligatured through an incision parallel with and just above the middle third of Poupart's ligament. This method is easy of performance, but the vessel is exposed and ligated very near the two branches, deep circumflex iliac and deep epigastric, upon which the collateral circulation depends. It is therefore not to be recommended.

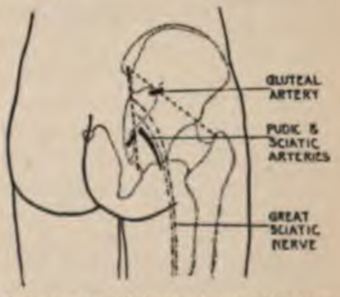
Collateral circulation after ligature of the external iliac artery. The internal mammary, lower intercostal and lumbar arteries, anastomose with the deep epigastric artery; the lumbar and lumbar arteries, circumflex iliac, internal circumflex with obturator and sciatic; external circumflex with gluteal perforating branches of profunda femoris, and external circumflex with sciatic pubic twig from obturator with pubic from deep epigastric.

LIGATURE OF THE INTERNAL ILIAC ARTERY.—This artery may demand ligature for hæmorrhage from, or aneurysm of, one of its larger branches (notably the gluteal artery when wounded, or the seat of an aneurysm near the great sacro-sciatic foramen. The artery is a short, thick vessel averaging one and a half inches in length, and dips downwards and backwards into the cavity of the true pelvis from the bifurcation of the common iliac artery to the upper margin of the great sacro-sciatic foramen. Anteriorly it is crossed by the ureter and covered by peritoneum; important posterior relations are the lumbo-sacral cord and the internal iliac vein.

Operation.—The artery may be reached by the same incision as that employed for the extra-peritoneal ligature of the external iliac artery, but this method is difficult and involves working in the dark. The intra-peritoneal operation is in all cases to be preferred. The incision four and a half inches in depth is made in the median line commencing one inch below the umbilicus. The intestines are drawn aside and the bifurcation of the common iliac artery, with the internal iliac artery springing from it, demonstrated. The peritoneum covering the artery is then incised and the artery cleaned three-quarters of an inch below the bifurcation of the common iliac. The aneurysm needle is passed from without outward, and care is taken to avoid including in the ligature the ureter.

LIGATURE OF THE GLUTEAL ARTERY.—This large branch of the internal iliac artery leaves the pelvis at the upper part of the great sacro-sciatic foramen above the pyriformis muscle. If a line be drawn from the posterior superior spine of the ilium to the centre of the trochanter major, the thigh being somewhat rotated inwards, the junction of the upper third with the middle third of this line will mark the place of emergence of the gluteal artery from the pelvis.

Operation.—A five-inch incision in this line exposes the fibres of the gluteus maximus muscle which are separated and retracted. The deep fascia beneath the great gluteal muscle is now divided, and the artery found at the upper edge of the pyriformis. The vessel is separated from its accompanying vein and the superior gluteal nerve, and ligatured as near to



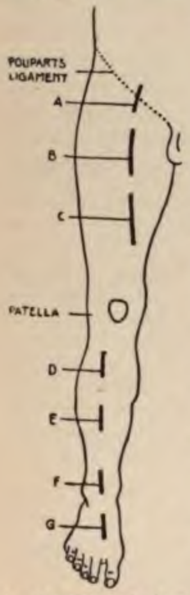
Posterior view of the hip showing the situation of the bones and chief arteries. (*After Quain.*)

the pelvis as possible on account of the division of the artery just after its emergence from the pelvis.

Ligature of the gluteal artery may be employed in cases of stab and traumatic aneurysm in which the artery may be presumed to be healthy. In the rare cases of spontaneous aneurysm, ligature of the internal iliac artery should be the operation selected.

Ligature of the Sciatic and Internal Pudic Arteries.—These vessels hardly ever demand the application of a ligature. If a line be drawn from the posterior inferior spine of the ilium to the tuberosity of the ischium, the junction of the middle third with the lower third of this line will mark with sufficient accuracy the point where the sciatic artery emerges from the great sacro-sciatic foramen from under cover of the pyriformis muscle. The internal pudic lies just internal to the sciatic artery.

Operation.—An incision in the line above mentioned, after separation of the fibres of the gluteus maximus and division of the deep fascia underneath the muscle, will expose the sciatic and internal pudic arteries. The vessels can easily be differentiated as the sciatic artery follows the course of the sciatic nerve, whilst the internal pudic artery, which lies to its inner side, re-enters and disappears through the small sacro-sciatic foramen. Either the sciatic or the internal pudic artery may be ligated, due care being taken to avoid any injury to their accompanying veins.



A, Ligature of Common Femoral Artery; B, Ligature of Femoral in Scarpa's triangle; C, Ligature of Femoral Artery in Hunter's Canal; D, Ligature of Anterior tibial artery in upper third of leg; E, Ligature of Anterior tibial artery in middle third; F, Ligature of Anterior tibial artery in lower third; G, Ligature of Dorsalis Pedis Artery.

Ligature of the Femoral Artery.—This artery is more frequently ligated for aneurysm than any other artery in the body. It frequently also demands ligature for hæmorrhage. The vessel may be tied in one of three places: (1) just below Poupart's ligament (the common femoral artery); (2) at the seat of election at the apex of Scarpa's triangle (superficial femoral); (3) In Hunter's canal. The vessel has therefore so great surgical importance that a short description of the course of the artery may here be given.

The femoral artery has its course in the upper three-fourths of the thigh, extending from Poupart's ligament above to the opening in the adductor magnus muscle, which it pierces to become the popliteal artery below. One and a half inches below Poupart's ligament, the artery frequently here called the common femoral trunk divides into the superficial femoral artery, which continues the course of the parent vessel, and the arteria profunda femoris, which is chiefly spent in supplying the muscles of the thigh.

The surface guide to the common and superficial femoral arteries is furnished by a line drawn from a point midway between the anterior superior spine of the ilium and the symphysis pubis to the adductor tubercle, the limb having been rotated outwards and abducted. In the upper four inches of its course the artery common femoral (above) and superficial femoral (below) lies in Scarpa's triangle, uncovered by any muscle, and here the vessel can be readily felt and compressed by the fingers. At the apex of Scarpa's triangle the sartorius muscle passing from without inwards overlies the vessel and continues superficial to it during the whole of the remainder of its course. Under cover of the sartorius the artery lies in Hunter's canal, in which also are contained the femoral vein and the internal saphenous nerve. The femoral vein is throughout closely applied to the artery. At Poupart's ligament the vein lies internal to the artery, then as the artery descends the vein passes in a backward direction and slightly outward, so that at the apex of Scarpa's triangle it lies behind the artery, and in Hunter's canal it is placed to its outer side. The deep femoral vein, one and a half to two inches below Poupart's ligament, crosses behind the artery.

Relation to Nerves.—In the groin the anterior crural nerve is placed external to the artery. Near the apex of Scarpa's triangle the internal cutaneous nerve crosses the artery, and in Hunter's canal the internal saphenous nerve lies in front of the vessel.

LIGATURE OF THE COMMON FEMORAL ARTERY is an operation very seldom performed, as, though the artery is easily accessible and readily tied, experience has proved that ligature of the external iliac artery is less dangerous. Secondary hæmorrhage and gangrene of the limb have occurred with remarkable frequency after ligation of the common femoral artery. For aneurism situated on the superficial femoral artery in its upper part, the external iliac is always to be tied in preference to ligature of the common femoral. For wound of the common femoral artery, however, in accordance with what has been above mentioned, the wounded artery itself must be doubly ligated, especially as ligature of the external iliac fails as a rule to arrest bleeding from the common femoral artery.

Operation.—A 3-inch incision in the course of the vessel is so made that one inch lies above Poupart's ligament, and two inches below it. The skin and superficial fascia are divided, and the lymphatic vessels and glands and the saphena vein and its tributaries are carefully avoided. Next the fascia lata is incised vertically, and the femoral sheath opened and the artery isolated as near to Poupart's ligament (*i.e.* as far above the profunda femoris branch) as possible. The vein lies to the inner side isolated from the artery by a septum of the femoral sheath. The aneurysm needle is therefore to be passed from within outwards, avoiding the crural branch of the genito-crural nerve which here lies in front of the artery. The artery may also be reached by an incision parallel to and half an inch below Poupart's ligament, but this method is inferior to the incision in the line of the artery.

LIGATURE OF THE SUPERFICIAL FEMORAL ARTERY.—This vessel may require ligation for aneurysm of the popliteal artery, for wound of the vessel, or for hæmorrhage lower down in the limb. In every case in which the surgeon can select the site of ligature, this is to be performed at the seat of election, viz. at the apex of Scarpa's triangle. This is one of the most successful ligations of large vessels in surgery.

Ligature of the Femoral Artery at the apex of Scarpa's triangle.—The thigh being everted and the knee and hip flexed, an incision, 3 inches in length, is made in the line of the vessel with its centre placed 4 inches below Poupart's ligament. The superficial and deep fasciæ are divided, and the inner margin of the sartorius muscle demonstrated as it crosses the lower half of the line of incision. The muscle is then drawn outwards, and the sheath is exposed and opened on its antero-external aspect, care being taken to avoid the long saphenous nerve. As the vein lies behind, and somewhat internal to, the artery, the aneurysm needle should be passed from within outwards, and great care must be taken to avoid injury to the vein, an accident of serious import and comparative frequency.

Ligature of the Femoral Artery in Hunter's Canal.—A 4-inch incision is made in the line of the vessel in the middle of the thigh. The skin, superficial fascia, and fascia lata are divided in the whole length of the wound, care being taken to avoid injury to the internal saphena vein, which, if seen, is to be drawn aside. The sartorius muscle which overlies the artery is recognised by the direction of its fibres (downwards and inwards), and is drawn inwards. The strong aponeurotic covering of Hunter's canal is exposed, and slit up with caution, the pulsation of the artery having been previously made out. The artery is now seen having the vein on its postero-external aspect, whilst the long saphenous nerve crosses superficial to the vessel from without inwards. The aneurysm needle is to be passed from without inwards.

Collateral circulation after ligation of superficial femoral artery.

1. Descending branch of external circumflex artery anastomoses with external articular twigs from popliteal artery.
2. Perforating branches of profunda with branches from popliteal.
3. Comes nervi ischiadici (from sciatic artery) with branches of popliteal.

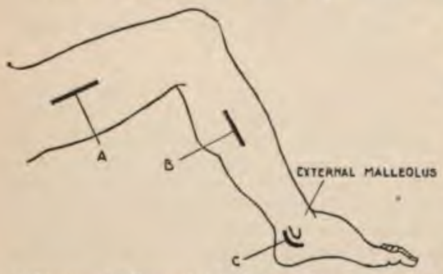
LIGATURE OF THE POPLITEAL ARTERY is an operation very rarely required. The artery extends from the opening in the adductor magnus muscle to the lower border of the popliteus muscle, where it divides into the anterior and posterior tibial arteries. The vessel at first courses downwards and somewhat outwards, lying upon the trigone of the femur, then reaching the back of the knee-joint it descends vertically upon the posterior ligament of the joint, and lower down upon the popliteus muscle. The vessel is deeply placed and is intimately attached to the popliteal vein, which is placed superficial to the artery, and lies on its outer aspect above and on its inner side below, thus crossing the artery from without inwards. The internal popliteal nerve is superficial to both vessels.

The popliteal artery may be ligatured just external to the tendon of the semi-membranosus muscle, or more easily in the lower part of the course. The former situation is the better.

Ligation of Popliteal Artery in its uppermost part.—A 4-inch incision is made immediately external to the outer margin of the semi-membranosus muscle and tendon, which are readily felt. The skin and fasciæ are divided when the semi-membranosus is seen, and retracted inwards. The fatty and areolar tissue is now separated and the internal popliteal nerve, exposed with the popliteal vein lying subjacent to it. These two structures must be drawn outwards after the vein has been most carefully freed from the subjacent artery. The aneurysm needle is to be passed from without inwards.

Ligation of Popliteal Artery in the lower part of its course.—A vertical incision 3½ inches long is made in the middle line of the popliteal space between the two heads of the gastrocnemius muscle. The skin and fasciæ are divided, the fat separated to either side, care being taken to avoid the external saphenous nerve and vein. The internal popliteal nerve and vein are now retracted outwards, and the aneurysm needle passed around the artery from without inwards.

THE POSTERIOR AND ANTERIOR TIBIAL ARTERIES rarely require ligation except for hæmorrhage from a wound of either vessel.



A, Ligature of Femoral in Hunter's canal; B, Ligature of Posterior Tibial in calf of leg; C, Ligature of Posterior Tibial behind outer ankle.

Ligature of Posterior Tibial Artery.—This vessel, the larger of the two terminal branches of the popliteal artery, reaches from the lower border of the popliteus muscle to a point midway between the internal malleolus and the internal tuberosity of the os calcis, where it terminates by dividing into the internal and external plantar arteries. The upper two-thirds of the artery lie under cover of the gastrocnemius and soleus muscles. In the lower third of its course, the vessel lies between the internal border of the tendo Achillis and the internal border of the tibia, covered only by skin, superficial and deep fasciæ. The artery has two venæ comites, and is crossed very obliquely from the inner side above to the outer side below by the posterior tibial nerve. The course of the vessel is indicated by a line drawn from the middle of the popliteal space to a point midway between the internal malleolus and the internal tuberosity of the os calcis. The artery may be ligated in the middle of the leg or behind the internal malleolus.

In the middle of the leg.—An incision 4 inches in length is made parallel to ¾ inch behind the inner border of the tibia. The skin and fasciæ are now divided, the internal saphenous vein and nerve being drawn aside if seen. The origin of the soleus muscle from the tibia is now divided, and the glistening aponeurosis on its deep surface having been identified, the soleus is drawn outwards, and the deep fascia lying between the soleus and tibialis posticus exposed and divided, when the artery will be felt pulsating with the posterior tibial nerve on its outer side. The artery is isolated from its companion veins, and the aneurysm needle passed from without inwards.

Ligation of the posterior tibial artery *behind the internal malleolus.*—A curved incision 2 inches long and concave towards the malleolus is made a finger's breadth behind the malleolus with its central point midway between the inner malleolus and the inner tuberosity of the os calcis. The skin, fascia, and internal annular ligament are divided and the artery found with a vena comes on each side, lying between the tendon of the flexor longus digitorum and the posterior tibial nerve (the tendon lying nearer to the internal malleolus). The veins are separated from the artery, and the aneurysm needle passed from without inwards. Care should be taken to avoid cutting into the tendon sheaths.

Ligature of the Peroneal Artery is very exceptionally required for injury. This vessel arises from the posterior tibial artery, about 1 inch below the bifurcation of the popliteal artery, and descends along the posterior aspect of the fibula.

Operation.—A 3½-inch incision is made over the posterior surface of the fibula, and the skin, fasciæ, and as far as may be necessary the fibular fibres of the soleus muscle are divided, and the muscle drawn inwards. The flexor longus hallucis is then seen, and the artery which lies under cover of this muscle is found by cutting vertically through the innermost muscular fibres close to the postero-internal border of the fibula. The artery lies with its venæ comites in a tube of tendinous structure.

Ligature of the Anterior Tibial Artery.—This is the smaller of the two tibial arteries. Leaving the popliteal trunk at the lower border of the popliteus muscle, it passes through the orifice in the interosseous membrane to reach the front of the leg. In the upper two-thirds of its course it lies deeply between the tibialis anticus on the inner side, and the extensor longus digitorum and extensor longus hallucis externally. In the lower third of its course, where the muscles have given place to tendons, the artery is more superficial, lies upon the lower part of the tibia, and is crossed from without inwards by the tendon of the extensor longus hallucis. The vessel has two venæ comites. The anterior tibial nerve lies at first on the outer side of the artery, then for the greater part of its course the nerve is placed in front of the artery, whilst at the ankle it has again got to the outer side of the vessel.

The course of the vessel may be with sufficient accuracy indicated by a line drawn from a point midway between the outer tuberosity of the tibia and the head of the fibula to the mid-point of the anterior aspect of the ankle-joint.

The anterior tibial artery may require ligature for injury, and in such a case the surgeon would enlarge the wound and ligature each end of the divided vessel. It is customary, however, to describe ligature of the vessel in the upper, middle, and lower thirds of the leg, though these formal operations are scarcely ever called for.

Ligature in the upper third of leg.—A 3-inch incision in the line of the artery divides skin, superficial and deep fasciæ. The intermuscular space between the tibialis anticus on the inner side, and the extensor longus digitorum externally is now opened up by separating these two muscles, when the artery, accompanied by its veins, is found lying on the interosseous membrane with the anterior tibial nerve on its outer side. The aneurysm needle is to be passed from without inwards.

Ligature in the middle third of the leg.—A 3-inch incision is made in the line of the artery. The skin, superficial and deep fasciæ are divided, and the interspace between the tibialis anticus and extensor longus digitorum opened up by separating the two muscles. The extensor longus hallucis is now found lying between the above-mentioned muscles, and is drawn outwards when the artery with its companion veins is discovered lying upon the interosseous membrane, after the anterior tibial nerve, which here lies in front of the artery, has been displaced outwards. The needle is to be passed from without inwards.

Ligature in the lower third of the leg.—A 2-inch incision is made in the line of the artery, reaching to within ½ inch of the ankle-joint, dividing skin, superficial and deep fasciæ, and part of the annular ligament. By extension of the ankle-joint and flexion of the great toe, the tendon of the extensor longus hallucis is made prominent, and this forms the best guide to the artery, which generally lies under cover of the tendon. If the incision is made higher up, the vessel may lie internal to the tendon, *i.e.*, between it and the tibialis anticus tendon. If very low down the artery may be external to the tendon, *i.e.* between it and the extensor longus digitorum. The nerve lies to the outer side, and the aneurysm needle should be passed from without inwards. In this operation care should be taken to avoid opening the sheaths of the tendons, otherwise adhesions of the tendons to neighbouring tissues may result.

LIGATURE OF THE DORSALIS PEDIS ARTERY.—This vessel continues the course of the anterior tibial artery. It reaches from the centre of the front of the ankle-joint to the hinder end of the interosseous space between the first and second metatarsal bones, where it passes into the sole of the foot. In its course over the dorsum of the foot it lies parallel with, and just external to, the tendon of the extensor longus hallucis.

Operation.—An incision is made in the line of the artery just external to the tendon of the extensor longus hallucis, and the vessel is found deeper than would be imagined lying between the above-mentioned tendon and the innermost tendon of the extensor brevis digitorum. The nerve lies on its outer side, and venæ comites accompany the artery. The aneurysm needle is passed from without inwards.

Before leaving the subject of ligature of the arteries below the knee, it must again be enforced that these operations can scarcely ever be required in practice, as the treatment of hæmorrhage is not a formal ligature, as described above, but opening up the wound and securing the injured vessel with ligatures placed above and below the bleeeding point.

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Arthritis, Arthrectomy, and Arthrotomy. *See* JOINTS.

Arthrodesis. *See* DEFORMITIES.

Arthropathy (Osteo), Hypertrophic Pulmonary. *See* OSTEO-ARTHROPATHY.

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INTRODUCTORY.—To no operation in surgery does the old adage *respice finem* apply with greater force than to an amputation, for the surgeon has to consider the future of the patient and to fashion that form of stump which will prove most useful when an artificial limb has been adapted to it. Thought must also be given to the social condition of the patient, because what is best for a wealthy man is not necessarily most serviceable to a pauper or even to a person of only moderate means. The end to be attained after an amputation of the arm is widely different from that required after removal of the leg. The arm is capable of many and varied movements, and the hand is constantly in view; more complicated and delicate apparatus is required therefore in the arm, and it is a golden rule to make all operations upon the upper extremity as conservative as possible. The movements of the leg are coarser, and the limb is more hidden from view, but, as it usually has to bear heavier weights and rougher usage, a more rigid and less complicated apparatus is sufficient to fulfil its main purposes.

RULES FOR AMPUTATION.—The rules for amputation differ therefore from a prothetic standpoint in the arm and leg. The following points must be borne in mind in amputating through the lower limb:—First, that the stump be not too long lest it be unwieldy; secondly, that it be not too short, lest it either slip out of its socket or become unduly flexed; thirdly, that, if it be near the ankle or knee it is better to allow room for an artificial joint rather than to disarticulate. These conditions are fulfilled in the leg by the following amputations which enable an artificial limb to be fitted with the maximum advantage, and the least discomfort. Syme's amputation is to be preferred for all operations in which the tarsus has to be removed: in the leg an amputation should be performed as low as possible in the middle third, since this allows sufficient room for the mechanism of the ankle joint, and enables the extensor muscles to overcome the powerful flexors. The best amputation above the knee is that which divides the femur in an adult about four inches from the articular surface of the condyles, because a stronger and more lasting knee-joint can then be made, whilst the extensor muscles are not destroyed.

LOWER LIMB.—When a limb has been removed for injury the stump undergoes certain well-recognised changes. It diminishes in size from disuse immediately after the operation, and, when an artificial limb is first worn, it gets still smaller, the shrinking continuing perhaps for twelve or eighteen months, until finally, as full control over the artificial limb is gained, and its use becomes second nature to the patient, the muscles increase again in size. The permanent condition of the stump is not reached, therefore, until about two years after the amputation, but no stump should be fitted with an artificial limb until at least three months have elapsed after it is soundly healed.

ANKLE.—The apparatus required after a Syme's amputation, or one in which it has been found impossible to preserve the tread of the foot, takes its bearings from the tuberosities of the tibia, more from the inner than the outer, and from the end of the stump itself, though, if the stump be tender, or if the patient is obliged to walk much, it is better to have the same apparatus as for an amputation below the knee. It consists (Fig. 1) of a leathern sheath reaching as high as the tuberosities of the tibia, and laced down the front of the leg. An artificial foot, jointed at its centre, is attached to the sheath by lateral rods which, by a joint, allow of movement at the ankle. The foot is made of light woodwork, and there is a false tendo Achillis to take off the strain which falls upon the ankle-joint when the full weight of the body is borne upon it. The instrument-maker requires a plaster-cast of the stump as high as the knee, and he should be supplied with the following measurements: the circumference of the stump below the knee, at the calf, below the calf, and at the lower end of the stump; and in addition, the length of the sound limb from the knee-joint to the ground. He should also be furnished with an outline of the sound foot and a boot. The price varies from fifteen to five guineas, but it can be reduced by substituting a circular boot without any movement at the ankle (Fig. 2) for the artificial foot.

Leg.—In every amputation of the leg and thigh above a Syme's amputation the bearings of the artificial limb must be taken from the ischial tuberosity and the ramus of the pubes, as the sawn end of the bone is too tender to bear the weight of the body. A more elaborate and expensive appliance is therefore necessary. When the knee-joint is left the use of the stump is to govern the artificial knee-joint; to aid its mechanism in walking and to bend the artificial limb when the patient sits down. The tibia and fibula should therefore be sawn across about their middle, as a four-inch stump gives the best results. The apparatus used after an amputation below the knee consists (Fig. 3) of a sheath of leather or thin wood fitting the thigh accurately, and extending from the level of the tuber ischii, the great trochanter and the descending ramus of the pubes to within four or five inches of the knee. Two lateral steel rods jointed opposite the knee attach the artificial calf to the sheath. The calf is a thin sheath of light wood loosely encasing the stump of the leg, it is attached by an ankle-joint to a foot whose front part is made of stuffed leather, whilst the heel and back part are wooden. The measurements required are: the circumference at the top, middle, and lower third of the thigh; the circumference at the knee-joint, below the knee, round the calf, and below the calf; the length from the perinæum to the knee-joint, and from the knee-joint to the end of the stump; the length of the sound limb from the knee-joint to the ground; the circumference at the knee, below the knee, round the calf, below the calf, and over the ankle at the instep. An outline of the sound foot should also be sent, and a boot, stating whether the right or left leg has been amputated. The price varies from thirty guineas to five pounds.

In the public services, and where economy is a great object, the "box leg" (Fig. 4) is used after all amputations of the leg below the knee. The patient rests upon his knee with the stump projecting backwards, the apparatus being fixed to the body by two straps, one encircling the hips and the other passing round both arms of the box and the contained thigh. The pin portion of the leg can be jointed to prevent the inconvenience caused by its projection when the wearer is sitting down. The price is from two pounds to fifteen shillings.

Thigh.—The artificial limb used after amputation through the thigh

(Fig. 5) contains a knee as well as an ankle-joint, but otherwise it differs but little from that used after amputation through the leg. The extension of the knee is brought about by an elastic mechanism attached to the front of the thigh above, and to the artificial leg below, whilst the flexion of the joint in walking is brought about by the weight of the body. Both these forms of apparatus are secured to the body by a girdle or by a suspender passing over the opposite shoulder. There is a general tendency to make these limbs too heavy, but it is possible to make the most elaborate apparatus, so that it does not weigh more than four pounds, and is both strong and rigid. The measurements required are: round the stump as high as possible, as well as round its middle and end; the length of the stump and the distance from the end of the stump to the ground when the patient is standing up; the circumference of the sound limb at the knee, below the knee, round the calf, below the calf, and over the ankle at the instep; the length from the knee-joint to the ground. An outline of the sound foot and a boot should also be sent, and it should be stated whether the stump is on the right or the left side. The cost varies from thirty to fifteen pounds, but it may be lessened by doing away with the ankle-joint and substituting a pin for the foot, as in Fig. 2.

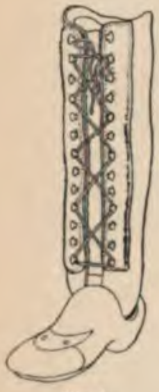


Fig. 1.

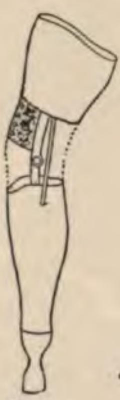
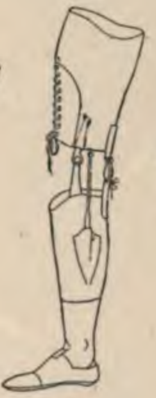


Fig. 2.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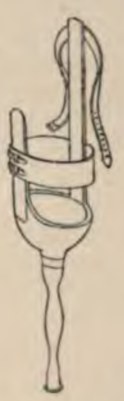
Fig. 4.

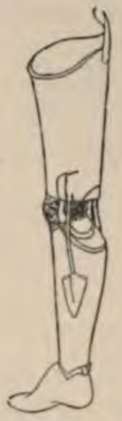
Fig. 5.

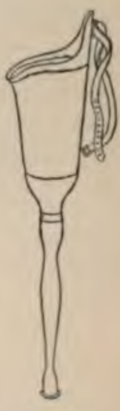
Fig. 6.

The service or "bucket leg" (Fig. 6) consists of a wooden sheath or bucket which receives the stump of the thigh, and into the lower end of which is fitted a "pin" upon which the patient walks. The pin is made of two parts, the one sliding within the other, and its long axis is directed a little outwards and backwards for greater ease in walking. The disadvantage caused by the pin projecting when the wearer sits, may be overcome by jointing it at the level of the knee, the joint being fixed or released by a spring catch. A bucket leg costs from eight guineas to one pound. It should be borne in mind that if a patient is going to wear an artificial leg he should not be allowed to learn to walk on a pin leg, as the method of progression in the two appliances is radically different.

The "Anglesea" leg is a wooden leg with hinge joints at the knee and ankle, the joints being brought into action by a long piece of catgut fixed at the back of the heel and ending at the knee. The catgut represents the flexor muscles of the knee and the extensors of the foot, whilst an india-rubber band fixed in front of the ankle and attached to a piece of wood in the centre of the calf counteracts the catgut cord and flexes the foot upon the leg. An india-rubber band is also fixed in front of the upper part of the leg to aid in impelling its lower portion forwards whilst walking.

The Palmer leg resembles the Anglesea leg, but the solid part of the leg is placed in a hollow portion of the foot, so that the limb looks neater, but is less strong.

The Bly leg is characterised by a ball and socket ankle-joint, with an arrangement of rubber springs and catgut cords to represent the more important muscles and tendons of the leg. The knee-joint also contains a mechanism representing the crucial ligaments, which lessens the shock conveyed to the stump in the act of walking.

Upper Limb.—The rules which apply to amputations of the leg by no means hold good for the arm. The mechanism of the artificial arm is of a prehensible character, and every care must be taken therefore to save as much of the natural limb as possible—in the hand, that the power of grasping may be retained, in the forearm to obtain pronation and supination, and in the arm to ensure flexion and extension as well as the rotatory movements of the shoulder.

Fingers.—A distal phalanx can be replaced, if necessary, by an apparatus fixed on like a thimble. All the fingers can be replaced by artificial fingers moulded on a cast modelled to fit the stump of the hand, the apparatus being laced over the wrist and concealed by a glove. When only a single finger has been removed at the metacarpo-phalangeal joint the deformity is usually so slight that it passes unnoticed, and no special apparatus is needed.

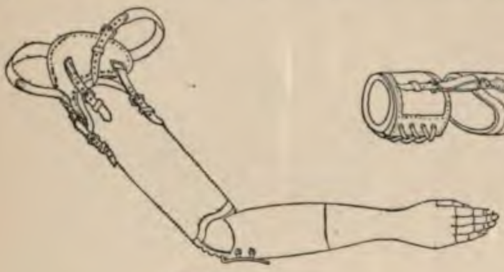

Fig. 8.

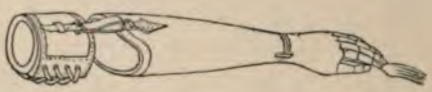

Fig. 7.

Hand.—When the whole hand has been amputated just above the wrist, an artificial hand can be adjusted to the stump with movable wrist and finger-joints. The wrist works so stiffly that it will remain fixed at any particular inclination, whilst the thumb is apposed to the index finger by a spring of sufficient strength to hold such light objects as a pen or a piece of paper.

Forearm.—After amputation through the forearm the stump is encased in a leathern sheath (Fig. 7) fitting it accurately, except at the upper end to allow of pronation and supination, and at the lower end where the cut extremities of the radius and ulna are usually too tender to permit of much pressure being exerted upon them. The sheath is continuous below with an artificial wrist, whilst above it is attached by straps or jointed steel rods to a band of leather or silk which encircles the arm just above the condyles of the humerus. The hand is removable at the will of the wearer, and can be easily replaced by a spoon, fork, hook, or such tools as may be of the greatest use to the individual. The measurements required are: the circumference above the elbow, at the elbow, and at the end of the stump: the length from the elbow-joint to the end of the stump; the length of the sound limb from the elbow to the wrist; a tracing of the hand with the fingers extended, stating whether the appliance is for the right or the left arm. The price varies from twenty guineas to five pounds.

UPPER ARM.—Almost the same apparatus is used when the arm has been amputated above the elbow, except that the circle of leather round the arm is elongated into a sheath extending to the shoulder above, and attached below by means of an elbow-joint to the representative of the forearm (Fig. 8). The apparatus is attached to the body of the patient by means of straps, and a very great amount of ingenuity has been displayed to obtain an arm which is capable of some of the various movements of the natural limb. The measurements required are: the circumference of the arm at the axilla, at the middle and at the end of the stump; the length of the stump from the axilla; the length of the sound limb from the axilla to the elbow, and from the elbow to the wrist; a tracing of the hand with the fingers extended should also be sent, and it should be stated whether the right or the left arm has been amputated. The cost varies from twenty guineas to five pounds.

The regulation appliances supplied by the Government to soldiers and sailors consist of the stump arm below the elbow, and the stump arm above the elbow. Neither of these appliances have elbow or wrist joints, but there are also Major Little's arms, known as "the improved Government arms," which have axes at these points allowing of limited movement.

The Beaufort arm was invented by the Count de Beaufort, who generously presented the original idea to the mechanician by whom it was constructed, on condition that its price should be within the means of the poorest patient. It is one in which, by a simple mechanism, the thumb and fingers remain shut so long as the natural elbow is bent, but on extending the arm and straightening the elbow the thumb expands, so that with a little practice it is quite easy to grasp even a small object.

LITERATURE.—HEATHER BIGG. *Orthropraxy; the Mechanical Treatment of Deformities, Debilities, and Deficiencies of the Human Frame.*

Artificial Respiration. *See* ASPHYXIA

Ascarides. *See* PARASITES.

Ascites.

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DEFINITION.—The word ascites does not denote any pathological process or disease. It is a convenient clinical term, which is applied to an effusion of fluid into the peritoneal cavity. The effusion may be due to obstruction of vessels or lymphatics, or it may be of inflammatory origin. The fluid may be serous, sero-fibrinous, hæmorrhagic, chylous, or milky, but collections of pus and extravasations of blood, bile, and urine are excluded from the common usage of the term.

Causation of Ascites in General.—Fluid may make its appearance in the peritoneal cavity, either (A) as the result of some local vascular affection, or (B) as a part of some general disorder.

(A) *Ascites due to local disease.*—(*a*) The most common cause of ascites is obstruction of the portal circulation. In this condition the blood-pressure in the peritoneal capillaries is raised and the transudation from them increases up to a point with which the absorption-power of the lymphatics can no longer cope. This obstruction may be situated (1) within the liver, or (2) in the trunk of the vein before it enters the liver.

(1) Within the liver the predominant cause is the compression exerted upon the small portal vessels by the contracting fibrous tissue of a multilobular (hob-nailed) cirrhosis. In the unilobular form of cirrhosis, ascites is uncommon, and, if present, is always small in amount. The rare infiltrating form of primary carcinoma of the liver may similarly produce ascites. Syphilis is a common cause, usually through the compression of larger portal branches by the cicatricial tissue, which is associated with caseating gummata. In such a case syphilis has usually been acquired. Less commonly the same changes result from the inherited disease. Inherited syphilis may also be a cause of ascites in young children by the development of a general interstitial hepatitis, but in such cases life is seldom prolonged. Any tumour, carcinoma, sarcoma, gumma, or hydatid may be so situated towards the under surface of the liver as to impinge upon the entering portal vein or one of its large branches. In rare instances, ascites has also been known to occur from the development of a tumour in such a position as to compress one or more of the hepatic veins at their junction with the inferior vena cava.

(2) Outside the liver the portal vein may be compressed by enlarged lymphatic glands in the portal fissure. This enlargement is usually due to a secondary carcinoma, and very rarely to any other cause, such as tubercle or lymphadenoma. A similar result may be produced by carcinoma invading the gastrohepatic omentum, by sarcoma, by carcinoma of the pancreas or duodenum, by a pancreatic cyst, or by an aneurysm in this region. Ascites may, in rare instances, ensue from the local perihepatitis which may be set up on the under surface of the liver by the presence of a new growth, gumma, or hydatid. More commonly it arises from the general perihepatitis which results from syphilis or is associated with chronic interstitial nephritis. In many such cases, however, the ascites is wholly, or in part, attributable to a coincident chronic peritonitis. Thrombosis of the portal vein is a further cause, but it usually occurs only in an advanced stage of cirrhosis or perihepatitis.

(*b*) Acute peritonitis from any cause is often attended with the effusion of fluid. This is commonly, however, small in amount, save when it is purulent in character, or when it is increased by the presence of bile, urine, or stomach-contents in the cases of rupture of gall-bladder, urinary bladder, or stomach respectively. An insignificant amount of fluid may also be thrown out in late stages of acute intestinal obstruction. On the other hand a considerable ascites is a common result of tuberculous or carcinomatous infection of the peritoneum. In the carcinomatous form the primary growth is more commonly situated in ovary or stomach than elsewhere. There is a marked tendency to peritoneal infection from a papilliferous tumour of the ovary. Sarcomatous infection of the peritoneum as a cause of ascites is much less common. A primary sarcoma may arise at any spot in the peritoneal or retro-peritoneal tissue, and in either case it may give rise to numerous scattered nodules over the whole peritoneal surface. A similar secondary infection may occur when the primary growth is elsewhere, more particularly when it is seated in testicle or ovary. A simple chronic peritonitis, which is sometimes associated with chronic interstitial nephritis, and is sometimes of unknown origin, is not uncommonly the cause of extreme ascites, necessitating repeated tappings. The mere presence of a tumour in the peritoneal cavity may be followed by the effusion of ascitic fluid, most commonly in the case of tumour of ovary or uterus. The enlarged spleen of leucocythæmia, and less commonly that of malaria, may act in the same way, but here the effusion may be partly attributable to the blood-condition.

(*c*) Under certain conditions ascitic fluid consists wholly or in part of chyle, derived from the thoracic duct or the lacteals. A chylous ascites is not a common event. It is usually moderate in amount, but as much as 16 litres of fluid may accumulate, and frequent tappings may be necessary. In rare cases the extravasation of chyle has been due to traumatic rupture of the thoracic duct in some part of its course. The duct has been ruptured by a carriage passing over the thorax, without apparent injury of pleura or lung. In one case rupture occurred during an attack of whooping-cough. But in most cases it results from obstruction situated either at some point of the duct, or in the mesentery, where the lacteals leave the intestine. Under the first head may be mentioned blocking of the duct by a secondary growth, carcinoma or sarcoma. It may be blocked by thrombosis at the point of junction of left subclavian and jugular veins. In one case these veins and the origin of the left innominate vein were found to be occluded by contraction. When the obstruction is thus high up in the course of the duct, a chylous effusion is commonly present also in one or both of the pleural cavities. Lower down in its course it may be compressed by a tumour of any kind, or it may be blocked by blood-clot resulting from injury. In one case the duct was found to be compressed by enlarged bronchial glands. It may be occluded by parent filariæ, but a chylous ascites is not among the common phenomena of filariasis. Under this head of duct-obstruction must be placed a recorded instance of mitral insufficiency and cardiac failure, in which the duct was found to be dilated, and chylous ascites ensued. In some of these instances of obstruction the duct ruptures, usually at the receptaculum, as is commonly the case after experimental ligation. Sometimes it is found obliterated, as if a previous rupture had occurred. Often it is dilated, and dilated lymphatics can be seen on the surface of intestine and mesentery. As regards the mesentery, the most common cause of a chylous ascites is a simple chronic adhesive peritonitis, whereby the lacteals are obstructed as they leave the intestine. The same result has been known to occur in tuberculous and carcinomatous peritonitis. Compression of the root of the mesentery by carcinomatous glands has also been noted as a cause.

(B) *Ascites as a part of a general disorder.*—Ascites commonly occurs as a part of the general dropsy of cardiac and renal disease. In both cases there is œdema elsewhere, and often also an effusion of fluid into some other serous cavity. Occasionally the ascites is considerable and is out of proportion to the dropsy of other parts. It may occur as the result of any form of cardiac failure, whether due to change in the cardiac muscle or to pericardial adhesions or to chronic pulmonary disease. Small amounts of fluid may be recognised also in conditions of extreme cachexia and anæmia from any cause.

CAUSATION OF ASCITES IN CHILDREN.—In children as in adults, ascites may be a part of a general dropsy of renal origin. It may also depend upon cardiac failure, whether due to valvular disease or to pericardial adhesions. On the other hand, chronic pulmonary disease and blood-conditions play little or no part in its production.

As regards local abdominal conditions, carcinoma as a cause disappears, cirrhosis sinks into the background, and tuberculous peritonitis is predominant. Cirrhosis has been noted at all ages, but, as a cause of ascites during the first five years of life, it is uncommon in comparison with tuberculosis of the peritoneum. A simple chronic peritonitis is even more rare than in adults. Sarcomatous infection of the peritoneum has been rarely recorded. Both sarcoma and enlarged tuberculous glands have been known to cause ascites by pressure on the portal vein outside the liver. A gumma or hydatid in the liver may be the cause, but with a syphilitic interstitial hepatitis, life is seldom prolonged beyond a few months from birth.

In the diagnosis of the cause of ascites in children assistance may often be obtained from the administration of an anæsthetic and from a rectal examination.

CHARACTERS OF ASCITIC FLUID.—Ascitic fluid varies considerably in its characters, and the following classes may be recognised:—

(1) SEROUS FLUID, resulting from portal obstruction, cardiac failure, and renal disease. It is usually clear, pale yellow or straw-coloured, with a slight greenish tint. It is occasionally opalescent, and the opalescence is not removed by filtration. It is alkaline or neutral. The specific gravity lies between 1008 and 1012. This maximum is sometimes slightly exceeded in ascites of cardiac origin, while the fluid of renal disease has commonly a lower specific gravity than that due to portal obstruction. It has very little power of spontaneous coagulation, but on standing a light clot will sometimes be formed. The proteid - percentage varies from 0·2 to 2·0, the lower limit being commonly associated with renal ascites, the higher limit with that of cardiac disease. In the amount and nature of salts, ascitic fluid resembles lymph. A trace of sugar is often present. Microscopical examination, after centrifugalising the fluid, may show a few leucocytes, red blood corpuscles, endothelial cells, and occasionally crystals of cholesterine. It is usually sterile.

(2) SERO-FIBRINOUS FLUID resulting from peritonitis, whether simple, tuberculous, or carcinomatous. In colour and appearance it often resembles the serous effusion, but it may acquire a reddish tint from admixture of blood. It is often clear. It may be opalescent, and in some cases it has been found to be milky, this appearance being due neither to the admixture of chyle nor to the presence of fat-globules, but to granules of globulin and nucleo-albumen. In the more acute inflammatory conditions it is apt to become turbid, owing to a great increase in the number of leucocytes. It differs from serous fluid in containing a larger amount of proteid, viz. from 2·5 to 6·0 per cent, in showing a greater tendency to spontaneous coagulation (the clot being often deposited in sheets or shreds among the abdominal contents), and in having a higher specific gravity, viz. from 1014 to 1024. No dependence can be placed on the appearance of cells, as distinctive of carcinoma, unless they are present in large numbers. Tubercle - bacilli are rarely found, but inoculation of a guinea-pig with the deposit obtained by centrifugalising affords a better chance of success.

(3) HÆMORRHAGIC FLUID.—A reddish tint may be acquired in cases of tuberculous or carcinomatous infection of the peritoneum. In rare cases, particularly in cirrhosis, less commonly with carcinoma, the ascitic fluid may come to resemble pure blood. In the cirrhotic cases a pleuritic effusion has also been found to be hæmorrhagic. It may be presumed that the blood is derived from the rupture of vessels in the peritoneum or in newly-formed adhesions.

(4) CHYLOUS FLUID is white and milky. Microscopic examination reveals the molecular basis of chyle, which consists chiefly of minute fatty particles, of which few are large enough to be recognised as fat-globules. The fat is in the form of an emulsion. A few lymphocytes may be found. It is alkaline, and of high specific gravity, about 1022. As compared with serous effusion the proteid-percentage is high, being between 2·0 and 4·5. The most striking difference, however, lies in the presence of fat, which amounts to 1·0 or 2·0 per cent, serous fluid, on the other hand, containing only a trace (0·04) of substances soluble in ether. A trace of sugar may be present.

(5) MILKY NON-CHYLOUS ASCITES (HYDROPS ADIPOSUS).—The milky appearance here is due to large and small fat-globules, and to large cells resembling colostrum-corpuscles. If allowed to stand in a tall glass, a creamy layer may form on the surface. It is occasionally met with in simple chronic, tuberculous, or carcinomatous peritonitis. It is due to the fatty degeneration of endothelial or carcinomatous cells, and it must be carefully distinguished from the true chylous form of effusion.

GENERAL EFFECTS OF ASCITES.—In extreme cases the fluid may amount to four or five gallons. Long before this extreme is reached a feeling of weight and discomfort is experienced, and the patient, if still able, walks with head and shoulders thrown back and legs wide apart. The tension of the fluid may be great, amounting to 25 or 30 mm. of mercury, and cases have been recorded where the umbilicus has given way. The fluid may distend the sac of a hernia. The skin of the abdomen is tense, smooth, and glistening, and lineæ albicantes are commonly present about the groins and flanks. Superficial veins may become visible, especially in the groins, irrespective of the cause of the ascites. From pressure on the vena cava, œdema of the lower extremities may result. To similar pressure on the renal veins may be attributed a diminution in the amount of urine and albuminuria. The tension in the abdomen serves to check the descent of the diaphragm. Breathing is carried on chiefly by the upper costal muscles, and is shallow. The heart may be displaced upwards, and irregularity of its action, or even faintness, may ensue. The basal parts of the lungs collapse, and the patient cannot breathe freely unless he is propped up in bed. Gastric and intestinal disorders are common, but are mainly attributable to the disease, of which the ascites is the result.

PHYSICAL SIGNS OF ASCITES.—Free fluid in the peritoneal cavity is to be recognised by enlargement of the abdomen, by substitution of dulness for the normal intestinal resonance, and by detection of a fluid "thrill" or wave. The abdomen becomes uniformly and symmetrically enlarged, with a tendency to bulge forwards and downwards when the patient is erect, but bulging laterally when he is recumbent. In extreme cases the lower ribs are pushed outwards, and the ensiform cartilage is upturned. In long-standing cases the umbilicus is often protruded.

The fluid, which is free to move in the peritoneal cavity, naturally accumulates in its lower parts, while the gas-containing stomach and intestine float upwards so far as their attachment will allow. In small effusions there may be only a little dulness in the flanks, which may reasonably be attributed to a loaded colon. In such a doubtful condition the fluid may be caused to gravitate towards the umbilicus, and to yield a dull note there, by placing the patient in the knee-elbow position. As the fluid increases in amount, dulness appears also above the symphysis pubis, at first perhaps only when the patient is sitting up. Later it is present also in the recumbent position, and it can be traced round into the flanks. The upper edge of the dull area is now crescentic, with the concavity directed upwards. The continued increase of the effusion is marked by the progressive shrinking of the resonant area lying within this crescentic outline. In extreme cases the resonant area is finally limited to a small oval or pyriform patch situated between the umbilicus and the ensiform cartilage. In rare cases, usually when the mesentery is shortened by chronic peritonitis, the resonant area is completely obliterated. In all degrees of ascites the boundary-line between the dull and resonant areas will be observed to shift as the patient turns from one side to the other, the resonant intestines tending to assume the uppermost position.

Over all the dull area in any stage of ascites, except perhaps when the fluid is confined to the flanks, a thrill or wave-shock can be recognised with one hand, if a sharp tap or fillip is applied elsewhere with the other. If the abdominal wall is thin, and the tension of the fluid is slight, the wave can even be seen. On the other hand, the thrill may be indistinct when the abdominal wall is fat or œdematous, and when the fluid is confined at a great pressure.

Finally, fluid may be recognised by vaginal examination, less easily per rectum. Douglas's pouch is depressed, the uterus is pushed down, and in extreme cases the posterior wall of the vagina has been known to present at the vulva.

Certain sources of error in the recognition of ascites are to be noted. Not uncommonly, even in cases of large effusion, a band of complete or modified resonance is present in one or both flanks. This is due to gaseous distension of the colon, and, in distinguishing between ascites and a large cystic tumour, it must be discounted. In acute peritonitis some quantity of fluid may be present, as shown by a thrill, and as proved by operation, yet the normal resonance is scarcely impaired. The fluid here is confined in the interstices between adherent and distended intestine, and it has little tendency to gravitate towards the lower parts, or to shift on change of the patient's position. Finally, a feeble thrill is sometimes obtained in the absence of ascites, when there is great tympanites or when the abdominal wall is very fat. Unlike a true fluid thrill, this may be prevented by the pressure of the edge of a book or piece of cardboard between the tapping and the receiving hand.

Ascitic fluid is said to be "encysted" when it is confined by adherent intestine, mesentery, or omentum. It is then difficult to detect, unless it be in considerable amount and in contact with the front abdominal wall. Such a collection may produce an unsymmetrical enlargement of the abdomen. It yields a fluid thrill and a dull note on percussion, but it closely resembles a cystic tumour in that the dull area does not shift on change of the patient's position.

DISTINCTION OF ASCITES FROM OTHER CONDITIONS.—(1) In the case of free fluid, the chief difficulty lies in distinguishing between ascites and a large thin-walled ovarian cyst. Abdominal examination alone may be insufficient to decide the question, but resonance in the flanks (with the reservation already mentioned), an unshifting character of the dulness, and any appearance of asymmetry of the abdomen (the distance between anterior superior spine and umbilicus being greater on one side than the other), or unequal resistance on palpation, are points to be looked for as indicating a cyst. Thrill, moreover, is seldom so distinct over a cyst as in ascites, and the greatest girth of the abdomen is usually below the umbilicus. When ascites coexists with an ovarian cyst, and when a cyst ruptures and discharges its contents, the difficulty is obviously increased. Vaginal examination, however, will reveal the close connection of a cyst with the uterus, which is commonly displaced to one side or to the front. Paracentesis should never be performed in such a case for the purpose of diagnosis, but the characters of ovarian fluid must be borne in mind. Some difficulty may arise in the case of general colloid carcinoma of the peritoneum. Here there may be considerable uniform enlargement of the abdomen, with complete dulness, but a true fluid thrill is feeble or absent. The distinction between ascites and obesity, œdema of the abdominal wall, and tympanites may be made from the physical signs already detailed.

(2) In the case of encysted ascites there may be great room for doubt, and many cases can only be cleared up by surgical exploration. A distended

bladder and a pregnant uterus may easily be excluded, if the possibility of their occurrence is remembered. A dilated stomach or gall-bladder and the conditions of hydronephrosis and pyonephrosis are usually easily recognised. A small ovarian cyst, and still more a parovarian cyst, may present great difficulty, and diagnosis must rest on the history of the case and on the result of a vaginal examination. Very rarely a cyst of the urachus, freely fluctuant, lying in the middle line above the symphysis, has attained dimensions which might lead to error. A pancreatic cyst lies above the umbilicus, mainly in the middle line, but sometimes more to one side than the other, having deep attachments, slightly movable, especially from above downwards, not moving with respiration. It may be impossible to distinguish it by physical signs from a collection of fluid in the lesser peritoneal sac. In many cases also exploration alone will serve to distinguish an encysted ascites from omental and mesenteric cysts, whether traumatic, sanguineous, serous, chylous, dermoid, or hydatid.

RECOGNITION OF THE CAUSE.—When ascites is a symptom of disease of heart, kidney, or blood, the diagnosis seldom presents any difficulty. When by exclusion of these causes it becomes clear that the ascites is due to some local abdominal disorder, a diagnosis is *often difficult and sometimes impossible.* In approaching the question it is well in the first instance to eliminate cases which present evidence of acute peritonitis. These cases are mainly of perforative origin, though occasionally a tuberculous peritonitis is so acute as to come into the class. In this class the abdomen is much distended, but this is due rather to tympanites than to ascites, and the fluid is usually small in amount, save when it is purulent, or when it is increased by the extravasation of stomach-contents, bile, or urine. In these cases the onset is sudden and severe. There is pyrexia, abdominal pain and tenderness, loss or impairment of the abdominal respiratory movement, thirst, vomiting, tendency to dryness of mouth and tongue, increased frequency of pulse, great constitutional disturbance and collapse. The picture is familiar and easily recognised.

As the next step, a decision must be made among the various forms of portal obstruction, and subacute and chronic peritonitis of simple, tuberculous, or carcinomatous origin. For this purpose there is required a systematic examination, based on a knowledge of the natural history of these several conditions. In some cases a certain diagnosis cannot be reached in an early stage. From the statistical side we learn the predominance of tuberculosis, cirrhosis, and carcinoma as the cause of ascites in early years, middle life, and old age respectively. But it must be remembered that cirrhosis may be the cause at any period of life, and in actual practice it is generally uppermost in the thoughts. On inquiry into the history of the patient, attention must be directed to such points as alcoholic excess, tuberculosis in the parents or other members of the family, syphilis (acquired or inherited), and previous symptoms pointing to new growth of stomach, bowel, ovary, or elsewhere, such as pain, vomiting, constipation, diarrhœa, hæmatemesis, melæna, and wasting. As regards the first point, it must be remembered that it is not very uncommon to meet with cases of cirrhosis, especially in young subjects, in which no suspicion of alcoholic excess can arise. The *mode of onset* must be closely examined, but in all the instances under consideration there is often the same gradual onset, and the history may reveal nothing more than a gradual failure of health and a steadily-increasing abdomen. A more acute onset points to tuberculosis.

From a *general view of the patient* further information can be gained. Jaundice, for example, is uncommon in the hob-nailed form of cirrhosis with which ascites is more particularly associated. It may be due to syphilitic affection of the liver. It is almost always present and intense with a primary carcinoma of duodenum, or head of the pancreas. It is generally present with carcinoma of the gall-bladder, and occurs in about 50 per cent of cases of secondary carcinoma of the liver, more especially when the glands in the portal fissure are affected. With a primary carcinoma of the liver ascites is found in about a third of the recorded cases, while jaundice is comparatively uncommon. This condition, however, is of such rarity that it hardly enters into a clinical consideration. The facial aspect may be of importance. In tuberculous peritonitis the face is apt to be pale, pinched, and drawn, while in cirrhosis it is commonly thin and sallow, with perhaps an icteric tinge of the conjunctivæ, and usually dilated capillaries on the cheeks. In carcinoma there is generally pallor, and the skin, in a late stage, becomes white and satiny. Cirrhosis and tuberculous peritonitis are commonly attended with wasting, but in neither case, except in late stages, is it so marked as in the case of carcinoma. Syphilitic affection of the liver, on the other hand, has little effect in this direction. The neck must be examined for tuberculous glands or scars, and evidence of tuberculosis in lungs, pleura, testicle, and elsewhere, must be sought for. Similarly the teeth, eyes, bones, and skin must be examined for indication of inherited syphilis. The inguinal glands may be enlarged in carcinoma of peritoneum. In tuberculous peritonitis the blood shows the changes of slight secondary anæmia without alteration in the number of leucocytes. There is but little alteration in the blood in cirrhosis, though in late stages there is often anæmia and a colour-index below par. This is the case also in an advanced stage of carcinoma, but here some degree of leucocytosis is often present, the number of white cells ranging between 10,000 and 30,000 per cubic millimetre (*vide* "Leucocytosis"). Though albuminuria may occur with extreme ascites of any origin, its continued presence points rather to cirrhosis or chronic peritonitis. Continued pyrexia is evidence in favour of tuberculous peritonitis; but it is not unknown in association with gumma or carcinoma of the liver.

In tuberculous peritonitis there is often some general abdominal pain and tenderness. This is not present in the other conditions, though occasionally in cirrhosis attacks of pain over liver or spleen may result from a local peritonitis. Superficial veins often become visible in all forms of ascites. Great enlargement of veins around the umbilicus, or between the umbilical level and the costal margin, points strongly to portal obstruction. Large accumulations of fluid are always suggestive of portal obstruction or simple chronic peritonitis, and in the latter case, owing to shortening of the mesentery, the whole surface of the abdomen may become dull. In tuberculous peritonitis the ascites is usually moderate in amount. In this affection, too, a distinctive redness and swelling are apt to appear around the umbilicus from time to time. Sometimes this is the precursor of a discharge of pus at this point. In carcinomatous infection of the peritoneum the umbilicus may become hardened by the subperitoneal deposit of growth at that spot. The liver may be felt in some cases of cirrhosis, either by a quick dipping movement of the fingers through the superjacent fluid, or more easily immediately after paracentesis. Hardness and perhaps granularity of the surface may be recognised. In cirrhosis the spleen may also be felt, if it is not obscured by the fluid, while this is rarely the case in tuberculous peritonitis. In most cases of cirrhosis, however, the edge of the liver is out of reach, except perhaps in the substernal notch. Gummata may be felt on the surface. In perihepatitis the liver is rarely enlarged. In the case of secondary carcinoma it is nearly always greatly enlarged, and nodules or tumours may be felt projecting from its surface. If such tumours are few and large, the possible presence of hydatids must be borne in mind. Finally, search must be made over the whole abdomen, and in the pelvis, for any solid mass. *The ovaries and the uterus must be carefully examined.* In many cases of tuberculous peritonitis, especially those of long standing, a mass of thickened omentum can be felt, sometimes vaguely, often distinctly, as a bar lying across the abdomen above or at the level of the umbilicus. An area of ill-defined resistance at any part of the abdomen may also be met with, due to omental thickening, or to the formation of intestinal adhesions. The same signs may be found when the peritonitis is of a simple nature, and sometimes the bowel is matted into a very palpable mass. In the case of carcinomatous infection of the peritoneum, hard and sharply-defined masses can usually be felt through the abdominal wall, and here again the infiltrated omentum can often be felt as a transverse bar-like tumour. But when the growth is in the form of disseminated miliary nodules, there is commonly extreme ascites and no solid material can be detected. Carcinomatous nodules, and tuberculous glands, or even caseous nodules on the peritoneum can occasionally be recognised on *rectal examination*.

After paracentesis *the characters of the fluid*, which have been already mentioned, more particularly as regards the specific gravity, proteid-percentage, and coagulability, will give important information as to the cause.

TREATMENT.—Ascites is a symptom, and the first step towards its treatment must be the recognition of the cause. The possibilities of removal of the fluid by medicinal action are limited. If it is due to cardiac or renal disease or to some general disorder, much may be done in this direction. Potassium iodide must be employed, if there be any possibility of dependence on syphilitic affection of the liver, but only in an early stage can benefit be expected. For the rest, medicinal treatment can only be of a general tonic nature, and any attempt to remove the fluid by purgation is not to be recommended.

The fluid must be removed by operative measures, whenever and as often as it comes to exert much pressure on heart or lungs. In the case of a tuberculous peritonitis the fluid must be removed through a small incision, according to the line of treatment laid down elsewhere, and an encysted ascites must be dealt with, if necessary, by incision and not by paracentesis. In all other cases simple paracentesis is sufficient. When the ascites is dependent on cardiac or renal disease, this is often unnecessary, but in cases of cirrhosis and chronic peritonitis repeated tappings are commonly required.

The patient should be placed in a semi-recumbent position, and it must be made certain that the bladder is empty. In order to maintain steady pressure on the abdomen during removal of the fluid, a many-tailed flannel bandage should be applied round the abdomen, so that the tails interlace behind. It should have an aperture cut in it, which will lie in the middle line below the umbilicus, and it should be broad enough to reach from the lower ribs to the symphysis. The skin should be cleansed at a point in the middle line midway between umbilicus and symphysis, and it may be rendered anæsthetic by a spray of ethyl chloride or by the application of a piece of ice dipped in salt. The skin and subcutaneous tissue must be punctured with a scalpel before the trochar is inserted, and both instruments must be previously sterilised by boiling. During withdrawal of the cannula the skin should be pinched up around it between finger and thumb. Having been rendered perfectly dry, the wound may be closed with a pad of lint or gauze soaked in collodion, the tails of the flannel bandage being finally brought round from behind and fastened together in front. Subsequent leakage occasionally occurs, especially if the fluid has been incompletely removed. This may be rectified by a second cleansing, drying, and closure of the wound.

Aseptic Treatment of Wounds.

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Introduction.—The word "aseptic" is an old one which has been revived. At first it was used without any scientific precision. Of late years, by a gradual process of evolution, it has been restricted to surgery, which aims at the absence of all bacteria from wounds. Within the recollection of even the present generation putrefaction was one of the ordinary concomitants of wounds. It was accompanied by a dreadful mortality from septicæmia, pyæmia, erysipelas, and various forms of gangrene. Continental surgeons (Reverdin) have said that nine-tenths of their cases of amputation through the thigh died of septic diseases; only operations of the direst necessity were undertaken. The interiors of the skull, thorax, abdomen, and of the articulations were forbidden ground. Gastrostomy was performed twenty-seven times with twenty-seven deaths. To Lord Lister belongs the fame of having recognised that putrefaction in wounds and their discharges was caused by living "ferments" (bacteria) and of having devised scientific methods of prevention. Various chemicals, especially carbolic acid and some of the salts of mercury, were brought into requisition, and the old term "antiseptic" was revived to designate them.

The use of antiseptics at once began to abate putrefaction in wounds, and at the same time brought about a remarkable diminution in septic diseases.

Several questions then arose with regard to pus and suppuration. *First*, whether the chemicals used in the treatment of wounds might not of themselves cause suppuration; and—

Secondly, whether suppuration was invariably caused by bacteria.

Even now it can hardly be said that these two questions have received a final answer. A full consideration of these questions will be found in the article "Suppuration." It will suffice to say now that aseptic surgeons accept the propositions (*a*) that the healthy unexposed tissues are sterile, and (*b*) that suppuration, erysipelas, pyæmia, septicæmia, and other traumatic diseases are caused by bacteria which enter from without. It follows that their aim is the *exclusion of all bacteria*. It would not suffice to exclude only those which caused decomposition, or those which caused suppuration; some, such as the bacillus of tetanus, cause neither, and the properties of others, especially of the skin bacteria, are unknown. Thus safety lies in the exclusion of all, or, in other words, in absolute sterility. Nor can any reliance be placed upon any form of immunity which the patient may be supposed to possess or have acquired. The exclusion of bacteria is within the surgeon's control; the patient's immunity is not.

To attain this result it is requisite to know (*a*) the sources of infection, (*b*) the steps required for the destruction of bacteria—disinfection, (*c*) the methods by which bacteria can be excluded during and after operations—antisepsis.

THE SOURCES OF WOUND INFECTION.—The commonest sources of wound infection are air, water, hands, and person of the surgeon and his assistants, instruments, utensils, ligatures, sponges, or materials brought in contact with the wound; the patient's skin or the patient's secretions, such as mucus, saliva, urine, or fæces. In practice it is assumed that all things are infected until they are known to be sterile. This rule eliminates much danger from instruments, sponges, ligatures, and appliances.

(*a*) *Air infection.*—The experiments of Professor Tyndall, Lord Lister, and others prove that the dust-laden atmosphere of cities, towns, and dwellings teems with all kinds of bacteria, and that the density of the dust particles is a rough index of their numbers.

The kinds of bacteria in the air depend to a large extent upon the origin of the dust. That which is blown from roads, fields, and gardens contains the spores of tetanus or of the Bacillus septicus (Nicolaier). Dust from ground which has been manured is full of the colon bacillus. In cities, owing to our irrational hygiene, the dust and dust-laden atmosphere contain tubercle bacilli (Marpmann). The air of hospitals contains pathogenic bacteria in considerable quantities. From the air of the operation theatre and wards of St. Bartholomew's Hospital we have grown, besides moulds and sarcinæ, Staphylococcus aureus, Staphylococcus albus, Streptococcus pyogenes, and various bacilli.

The atmospheric bacteria were thought at one time to be one of the chief sources of wound infection. Lord Lister endeavoured, by using a carbolic spray, to produce a germ-free atmosphere. But soon it was discovered that the carbolic acid in the spray was insufficient to kill the atmospheric bacteria. Further, there was reason to suppose that the spray actually carried additional bacteria into wounds. After this, to protect from air infection, screens of antiseptic gauze wet with antiseptic lotion were used to cover wounds. The danger of air infection is at the present time rather underrated. It may, however, be much diminished by precautions. Tyndall's experiments conclusively proved that when the atmosphere was still, dust and bacteria gravitated and left a pure air. Therefore it is inadvisable to operate in theatres or rooms which have just been disturbed. Next, the theatres, wards, and corridors of hospitals should be planned with smooth surfaces and without ledges or grooves where dust can accumulate. As yet British surgeons have not had screens of glass erected betwixt the audience and the floor of the operation theatre, nor do they consider it necessary to wear a mask (Hübner). Operations should be done as swiftly as efficiency will allow. Screens of antiseptic gauze should be used to protect one part of the wound whilst another is being dissected—as, for instance, in amputation of the breast—to protect the pectoral region with a layer of antiseptic gauze during the dissection of the axilla, or the upper part of a laparotomy wound during manipulations within the pelvis. Inasmuch as bacteria undoubtedly fall from the air into wounds, most surgeons follow Lord Lister's practice and at intervals rinse them out with antiseptic lotion. This device is objected to by some because the tissues are supposed to be injured by the chemical, their reparative power impaired, and rendered less resistant to infection; therefore they endeavour to evict them with copious ablutions of sterilised water or sterilised salt solution. In my own surgical practice solutions of biniodide of mercury (mercuric iodide) are used. These are made by dissolving red iodide of mercury in water with the help of about an equal weight of iodide of potassium. The strength of the lotion is determined by the conditions. For most cases the standard strength of 1 part in 2000 parts of water is used, for abdominal operations 1 part in 4000. This salt of mercury is a highly efficient disinfectant. Its strength is not less than twice that of sublimate. It is unirritating and does not combine with albumen. Cut surfaces to which it has been applied remain quite fresh and red and do not assume the pickled appearance which follows the application of carbolic acid or sublimate. As it all runs out again it seldom causes mercurial poisoning. Instead of lotions, swabs of sterilised wool and gauze are sometimes used to cleanse wounds or cavities. This saves the cells from being swollen or burst by the imbibition of the fluid, and from being poisoned by the chemical.

(*b*) *Water infection.*—The water from taps and ordinary sources contains a great variety of bacteria. Amongst the pathogenic are *Staphylococcus pyogenes aureus*, *Streptococcus pyogenes*, the bacillus of *diphtheria*, the *colon bacillus*, and many others; the non-pathogenic are too numerous to mention. Amongst the latter are many saprophytes. The latter are harmless under ordinary circumstances, but can flourish and cause the most harmful effects when placed under favourable conditions, as, for instance, when they are introduced into a peritoneal blood-clot, or into a pleuritic effusion. Aseptic surgery guards against such occurrences because its aim is the exclusion of all bacteria. A proper supply of sterilised water is therefore necessary. The ordinary hot-water supply is not dependable. On more than one occasion we have grown bacteria from water supplied to operation theatres from distant boilers. I have not tested the hot-water supply of private houses. It is, however, very easy to sterilise water by keeping it at boiling-point for thirty minutes in a suitably covered vessel.

Five minutes in boiling water kills most bacteria, but the longer period is needed to ensure the death of those which possess spores. Before an operation a supply of water should be sterilised and allowed to cool. During the cooling the vessel should be covered and protected against the entrance of dust—by a closely-fitting lid, or a thick cap of antiseptic gauze over the mouth and spout.

In addition to heat, *filtration* and chemicals are available for the sterilisation of water. Filtration has hitherto been little used. Sterilising can be ensured by using the Pasteur-Chamberland filter. The process is slow and requires much vigilance to ensure reliable results.

The *addition of chemicals* to water which has been boiled is an additional guarantee of its sterility. Such chemicals as sublimate, biniodide of mercury, cyanide of mercury, several other metallic salts, carbolic acid, sulphurous acid, chlorine, permanganate of potash, and so forth not only afford solutions which are sterile, but also lotions with disinfectant and antiseptic properties.

The mercuric salts are sufficiently powerful to sterilise ordinary tap water in a few minutes. The proportion of chemical used in ordinary surgical work is sufficient. Carbolic acid is also efficient, but considerable quantities of it are required—1 to 5 parts in 100. Water is exceedingly easy to disinfect, because it contains no substance to combine with the chemical and render it inert.

(*c*) *Contact infection.—The hands.*—Until recently a great deal of wound infection must have been caused by the fingers of the surgeon or of his assistants. Even with our present improved methods the disinfection of the hands is by no means easy. Something depends upon the quality of the skin. That which is smooth and thin is easier to sterilise than that which is rough and fissured.

The bacteria of the skin reside in the ducts of the sweat-glands, in the sebaceous glands, and in the mouths of the hair follicles. The sebaceous material which all parts of the human skin contain is one of the chief impediments to the action of disinfectants. The skin of the hands is less greasy than that of the scalp, back, or trunk, but, nevertheless, its disinfection requires considerable skill and experience.

The chinks and crannies about the nails are an additional danger. Various bacteria have been grown from the skin of the hands. Staphylococcus albus and aureus, Streptococcus pyogenes, Bacillus pyocyaneus, the colon bacillus, and many others have been found. Leedham-Green grew many kinds of bacteria from hands which had been scrubbed and washed for fifteen minutes in hot water and soap.

The hands of those who touch septic wounds, or who perform post-mortem examinations, may carry the germs of virulent and deadly diseases.

DISINFECTION—*Of hands of surgeon and his assistants.*—The hands are prepared for disinfection by divesting them of rings. This injunction is needless for the modern school of aseptic surgeons who contemn such trivialities. It is safest to trim the nails as short as possible. Those who object to this may try to cleanse the nails with 10 per cent iodoform gauze and 5 per cent carbolic lotion (Frantz Boll). The gauze has to be pushed along beneath the edge of the nail. Assuming that the nails have been trimmed as close as possible, the next step in the preparation of the hands for disinfection is the removal of all visible dirt and all greasy material by the energetic use of a sterilised nail-brush and hot water and soap. We have had successful results with all kinds of soap, but the experiments of Koch and of Boll show that *potash soap* (soft soap) is rather more efficacious than hard soap.

Ether and turpentine have also been used at this stage for the more complete removal of fat. *Turpentine* is the best, because it possesses slight germicidal properties. Their use, however, tends to roughen the skin, and solutions of chemicals in spirit renders them unnecessary.

After the hands have been cleansed of dirt and grease, various chemicals are used as disinfectants. The thoroughness with which the disinfectant is used is the most important point. Many are unable to endure the strong mixtures of carbolic acid and perchloride of mercury which some surgeons find efficacious (Watson Cheyne). It is now generally believed that *alcoholic applications* have the most penetrating properties and give the best results (Poten). The hands may be soaked for one minute in 80 per cent alcohol, and then for one minute in 2 per cent sublimate lotion (Fürbringer).

I myself have obtained the best results with solutions of biniodide of mercury in methylated spirit. This is made in the following manner:—First, a strong solution (1 in 10) of mercuric iodine is prepared. This serves also for making lotions.

Take of

Mercuric iodide	100 grms. or 960 grs.
Iodide of sodium or potassium	100 grms. or 960 grs.
Distilled water	1000 c.c. or 20 fl. oz.

The iodide of potassium or sodium is dissolved in the distilled water and the mercuric iodide is added. If the latter is impure and contains mercurous iodide a green precipitate results. Otherwise a perfectly clear solution is obtained.

The spiritous solution is made as follows (Parsons):—

Take of

1 in 10 solution of biniodide	10 c.c. or 192 minims.
Methylated or rectified spirit	375 c.c. or 15 fl. oz.
Distilled water to make final product	500 c.c. or 20 fl. oz.

The percentage of water is added because Koch's experiments showed that solutions of chemicals in absolute alcohol are inert. Methylated spirit is used because it is cheap.

The hands and forearms are soaked in this 1 in 500 solution of biniodide of mercury for not less than two minutes *by the clock*. Three of my house-surgeons disinfected their hands by this method thirty-eight times out of thirty-nine. It is now unusual to have a septic result when the skin is tested before the operation. Before and during operations the disinfected hands are frequently rinsed in lotion of biniodide of mercury, 1 in 2000.

A small, clean, non-suppurating cut upon the hands may be covered with a small collodion dressing. It is unjustifiable for a surgeon to operate with any kind of suppurating sore upon the hands or fingers. I have known the most painful results follow such a proceeding.

Disinfection of the skin of the patient.—The difficulty of disinfecting the patient's skin depends mainly upon the size and depth of the sebaceous and sweat glands. The skin of the neck, limbs, and abdomen can be disinfected with ease; whilst that of the back, groin, scrotum, and scalp is exceedingly difficult. I cannot ascertain that the last has ever yet been disinfected.

The process of disinfecting the patient's skin is almost exactly the same as that which is used for the hands of the surgeon and his assistants; but as the difficulties are greater, rather more elaborate precautions are necessary. *First*, the hair is shaved over the whole area of the field of operation, or over that which is covered by the dressing. Sometimes the shaving is done in such a perfunctory manner that half the pubic hair is left, although an operation is about to be done for the cure of hernia or of varicocele. *Second*, as much as possible of the sebaceous material and epidermal scales are thoroughly removed by energetic scrubbing with hot water and soap. *Third*, the remaining fat is extracted with turpentine; a variety of other chemicals may be used for this purpose. Ether, alcohol, solutions of soap in alcohol, and benzine have been used and given good results.

After the extraction of the fat the skin is disinfected with strong solutions of sublimate (1 in 500), of carbolic acid (1 in 20), or of both together, of formalin (1 per cent solution—Landerer and Krämer), or of chemicals in spiritous solutions. My best results have been obtained with the 1 in 500 solution of biniodide of mercury, which is used for disinfecting the hands. This has to be soaked in for not less than two minutes by the clock. In hospital practice the patient's skin is usually disinfected several hours before the operation. It is therefore necessary to protect it with an efficient antiseptic dressing. Further, the precaution is taken to repeat the disinfection with spirit and biniodide of mercury before proceeding with the operation.

Such rigorous methods of disinfection cannot be used for *parts lined with mucous membrane*. Before operations upon the rectum the risks of sepsis are diminished by emptying the bowel with copious enemata, and washing away mucus or discharge with solutions of boric acid. Stronger antiseptics are used just before the operation (*e.g.* 1 in 2000 biniodide of mercury lotion). Sometimes a preliminary inguinal colotomy may be required.

Before vaginal operations the external genitals and perineum are thoroughly scrubbed and cleansed with soap and water. The vagina is cleansed with soap and water, with the help of absorbent wool (Robb), and disinfected by abundant douching with an antiseptic, such as warm biniodide of mercury lotion, 1 in 2000.

Before performing urethral operations I have of late endeavoured to douch the urethra with large quantities of 1 in 2000 biniodide of mercury lotion. Should the urine be septic, the bladder is washed out with dilute solutions of nitrate of silver (1 gr. in 1 oz.).

The conjunctiva can only bear the gentlest treatment. M'Gillivray recommends that the palpebral aperture and tarsal folds should be irrigated with sterilised salt solution at a temperature of 90° F.

Disinfection of sponges.—Although marine sponges possess undoubted advantages, some surgeons have discarded them because they are difficult to disinfect. Their place is taken by swabs of cotton-wool wrapped in gauze and sterilised in the autoclave. The asepticity of these is undoubted, but they do not clean a wound of blood so rapidly as sponges; also, wool is apt to be left in the wound should it contain rough surfaces of bone. In abdominal operations swabs of wool require minute supervision to prevent any being lost. However, the following method of disinfecting sponges gives perfect results, when carried out by an intelligent and conscientious person. If the sponges be new, (1) the sand is shaken and beaten out. (2) Shell and coral are dissolved by soaking for twenty-four hours in a solution of sulphuric or hydrochloric acid (1 drachm of strong acid to 1 pint of water; the mixture requires to be made with extreme care to avoid accidents. (3) The acid is removed as far as possible by squeezing in warm water, and the sponges are then soaked for half an hour to an hour in a warm solution of washing-soda (ʒj. to 1 pint of water). This removes fat and albumen. The process may require to be repeated several times. (4) The soda solution is washed out with warm sterilised water, and the sponges immersed in cold solution of sulphurous acid (1 in 5) for twelve hours, for a final bleaching and sterilisation. Sponges prepared by this method were sterile forty times out of forty-one. The contamination probably came from the air.

Disinfection of utensils and instruments.—Heat is the simplest and most reliable method of disinfecting the utensils, instruments, and materials required for an operation. Dry heat is hardly practicable. To disinfect glass test-tubes and culture dishes, a temperature of 180° C. is required, and has to be maintained for not less than thirty minutes. At this temperature the vessels become red-hot, and most instruments would be ruined. The perfect reliability of moist heat in the form of steam or boiling water has now been abundantly proved by the work of Koch, Klein, Parsons, Delépine, Reid, and of many other workers. But the action of steam cannot be relied upon unless it is applied for some hours or used under pressure. Steam at a pressure of 10 lbs. kills the most resistant spores in thirty minutes. *A special apparatus* is required, and at St. Bartholomew's Hospital the utensils, wool, gloves, and towels are disinfected in a steam autoclave of the *Lantenschläger* pattern. The towels were sterile in thirty-eight tests out of forty. One of the infected towels contained hay bacillus, and the other an unascertained bacterium. The infection probably came from the air in both instances.

It is, however, impracticable in surgical practice to use steam on every occasion. Therefore, boiling water, or water to which 1 per cent of washing-soda has been added, is used for towels, utensils, drainage-tubes, and instruments. The washing-soda (1 drachm to the pint) removes grease and albumen, and by raising the temperature of the water renders it a most efficient disinfectant. It also helps to prevent oxidation. An immersion of thirty minutes is required to ensure sterility. Boiling oil and glycerine have also been used for the disinfection of instruments (Miguel and Tripier). Boiling water and washing-soda can be obtained in every dwelling. Instruments endure a great deal of boiling if they are properly plated with nickel. Knives must have metal handles, and the blades should be protected lest they become gapped and blunted during ebullition. It is safer not to use the same instruments for more than one operation. After use they ought to be thoroughly scrubbed with soap and water, ready for disinfection. It is convenient to have scissors and pressure forceps with joints which allow the two halves to come apart. I do not use bayonet-jointed instruments myself, because I have often seen them come apart during operations, and within the abdomen.

The aseptic instruments are placed at once in 2 per cent carbolic lotion. The surgeon takes them out of this, uses them, and puts them back again. It is a sign of ineptitude when instruments are promiscuously squandered over towels, undisinfected garments, or tables.

For emergency operations, the utensils may be scrubbed with soap and water, and flamed with alcohol.

The perfection of some instruments would be greatly impaired by the violent expansion and contraction caused by boiling. Lithotrites, urethrameters, urethratomes, endoscopes should be washed and scrubbed scrupulously clean with hot water and soap, and the various parts thoroughly wiped over with a little pure carbolic acid, or immersed in a dish of pure carbolic acid. This may require to be slightly warmed, or mixed with a small proportion of water or glycerine to keep it liquid. The carbolic acid is washed off with 2 per cent carbolic lotion, and the instrument dried with a sterilised towel, and oiled with a little sterilised olive oil.

Catheters and sounds should be made of material which can be sterilised by heat. Olivary bougies, bullet-headed bougies, and gum-elastic catheters would be destroyed by such treatment. All danger of infection is obviated by using *new* instruments with perfectly smooth and polished surfaces. These should be soaked in sublimate or biniodide of mercury lotion, and lubricated with glycerine or sterilised oil. It is not necessary to add chemicals to oil or glycerine, because the mixture is inert.

The instruments used in ophthalmic surgery require most delicate handling. M'Gillivray says that new instruments, and instruments that have been used for aseptic cases, bear boiling for two and a half minutes in 1 per cent solution of soda. They are then dried with a sterilised cloth and stored in aseptic boxes in an air-tight cabinet. Before use they are again immersed for two and a half minutes in boiling water. This amounts to Tyndall's "*intermittent sterilisation.*" Bacteriological tests show that instruments treated in this way are sterile.

Hypodermic syringes ought to be made with asbestos or metal plugs, so as to be disinfected either by dry heat or by boiling. Usually the needle and syringe are boiled in a test-tube. Hypodermic solutions ought to be sterile. They are safest when made with tabloids and sterilised water. The skin ought to be disinfected with biniodide of mercury and spirit lotion (1 in 500) before the injection. This applies to subcutaneous, intravenous, and intramuscular injections.

Disinfection of ligatures and sutures.—Twisted or plaited silk are favourite materials for sutures. The stoving and bleaching which the silk undergoes in the process of manufacture are well calculated to render it sterile. Absolute certainty is easily attained by boiling the silk in water for not less than twenty minutes. It is convenient to wind the silk upon a glass reel, and care should be taken not to have too great a bulk, lest the heat fail to penetrate. It is not necessary to use carbolic lotion. The carbolic acid is merely evaporated by the heat. House-surgeons and others often commit the blunder of putting the silk into the soda solution along with the instruments. The alkali removes some part of the silk and renders it rotten. After the silk has been sterilised it is kept in 5 per cent carbolic lotion, and never touched except with sterilised fingers or instruments. Silk treated in this manner was sterile on forty consecutive occasions.

Silkworm gut or *fishing-gut* is peculiarly well adapted for suturing wounds. It is especially useful in operations for the repair of hare-lip, cleft-palate, or ruptured perineum. It bears heat well, and is only made more supple and tough by soaking in carbolic lotion. It is therefore sterilised the same as twisted silk by being boiled in *water* for not less than twenty minutes, being subsequently kept in 5 per cent carbolic lotion. Fishing-gut treated in this manner was sterile thirty-five times in thirty-seven. It is clearly rather difficult to disinfect.

Catgut is very difficult to disinfect and rather unreliable. I have seen recurrent hæmorrhage more than once in cases in which it has been used for ligatures. But for circumcision, plastic operations in children, and in wounds already septic it has undoubted advantages. It is not desirable to place silk ligatures on the stump of the appendix or upon the meso-appendix in a case of perforative or gangrenous appendicitis. It is also a safer material for closing rents or wounds in the urinary bladder. It is now clearly understood that the great bar to the disinfection of catgut is the grease which the makers rub upon it to give a smooth and shining appearance (Reverdin). The first and most essential step, therefore, is the removal of this grease by winding the raw catgut upon a board, scrubbing with hot water and soap for half an hour, and then immersing for twenty-four hours in ether. When no more fat can be extracted the chemical disinfectant can be applied. I am at present using a 1 in 250 solution of biniodide of mercury in water. The catgut soaks in this for seventy-two hours, after which it is tested and used. It is kept in the same solution, and is quite strong and good at the end of a year. A great variety of disinfectants have been used. Sublimate in alcohol 1 per cent, sublimate 1 in 1000, followed by Juniper oil and absolute alcohol. Heat in various forms has been tried—boiling alcohol (Robb) and dry heat (Reverdin, Braatz). It is possible that the complexity of these methods has prevented them from being much used.

Little is known concerning the disinfection of the *other animal ligatures*, such as kangaroo tendon, rat-tail tendon, or gold-beaters' skin. It would therefore be well for any one who thought of using them to test their asepticity by immersion in culture broth.

Aseptic and Antiseptic Dressings.—A great many dressings are now upon the market. Some surgeons use gauze which has been sterilised by heat. This is quite unirritating, but inasmuch as the disinfection of the skin is still uncertain it seems advisable that the gauze should contain a store of chemical antiseptic, such as carbolic acid, sal alembroth, cyanide of mercury, iodoform, and so forth. We have obtained the best results with 5 per cent carbolic gauze, 2 per cent sal alembroth wool, and 10 per cent iodoform gauze. The mercuric preparations seem liable to cause dermatitis when long in contact with the skin; nevertheless, they give excellent results when properly used. Other points will be mentioned when the final dressing of the wound is described.

The Field of Operation.—For prolonged operations a *hot-water table* or a water-bed ought to be provided. One of the cheapest, simplest, and best hot-water tables is that which is called the "Swiss pattern," and was devised by Dr. Jussaud. The position of the patient is arranged so that no further movement or disturbance is required, and so that all fluids can run away clear of the patient's body.

The bandages and protective dressing are then removed from the disinfected area. The garments are covered with thin mackintosh or dry towels, and the field of operation prepared by arranging sterilised towels all round to exclude all that is not known to have been disinfected. In operations about the lower part of the abdomen, scrotum, or groin, the organs of generation are wrapped in antiseptic gauze wrung out in lotion.

Near the right hand of the surgeon a table is placed and the dish of instruments; the bowl with sutures and ligatures, and a basin of lotion for the hands, are arranged upon it. This table is completely covered with sterilised towels. The operation is done cleanly, neatly, and expeditiously, and with as little bruising or handling as possible. The surgeon takes the knife or other instrument from the lotion, and after use replaces it in the lotion. Bleeding is stopped at once with pressure forceps. Loss of blood is attended with many evils, and vessels allowed to bleed are apt to infiltrate loose tissues with blood. At the end of the operation ligatures should be applied until the wound is dry. Too much care cannot be lavished in closing the wound. Cavities should be obliterated with buried sutures.

This is of great value after the removal of tumours of the thyroid body, and after the excision of cysts or innocent growths from the mammary gland.

Drainage is now provided in all septic cases. Our present methods of disinfection with chemicals cannot be relied upon to disinfect a septic wound or cavity. In aseptic wounds drainage has sometimes to be provided to allow blood to escape, especially where a large area of cancellous bone is exposed, as in Syme's amputation, or amputation through the condyles of the femur. Also a drain may be needed where the tissues are very loose, and in which recurrent bleeding is apt to occur. For this reason I drain all scrotal wounds after exploration of the testicle, castration, radical cure of hydrocele or varicocele. A drain may also be required when inflammatory effusion is expected. A drainage-tube in the knee-joint, after wiring the patella, vastly diminishes the subsequent pain. A drain of india-rubber tubing or gauze is now oftenest used. In abdominal surgery the use of glass tubes is ceasing, because rubber tubes are found to answer as well, and to cause less pain. Glass tubes have been known to break.

IRRIGATION.—Since the abandonment of the spray it has been customary to apply large quantities of lotion at intervals during the operation, with the idea of washing out any bacteria that may have been introduced or fallen from the atmosphere, and also with the remote possibility of killing them. It is probable that irrigation has been somewhat abused. The application of strong solutions of carbolic acid or of sublimate must be harmful. Wounds treated in such a way look as if they had been pickled. The tissues become covered with INSOLUBLE ALBUMINOUS COMPOUNDS, and afterwards inflame and secrete blood-stained serum. They are also rendered much less resistant to infection. Irrigation should therefore be used in moderation. A 1 in 2000 to 1 in 4000 solution of biniodide of mercury forms no combination with the tissues, and effectually removes blood or bits of fat. Its specific gravity is greater than normal saline solution, so that the tissue cells are not burst. Biniodide of mercury causes signs of poisoning if applied in quantity to large wounds, or if left in the chest or abdomen. Therefore, in amputation of the breast or laparotomy, the weak solution, 1 in 4000 or 1 in 5000, should be used. Biniodide of mercury has twice the germicidal value of sublimate, and is less poisonous. Large cavities like the abdomen ought to be irrigated thoroughly, or not at all. If the sepsis is local, it is better to trust to swabbing. There is always a danger of the irrigation fluid washing septic materials into places whence it may not be dislodged, as, for example, behind the liver in cases of perforation of the stomach or duodenum.

THE DRESSING.—When the wound has been sutured it has to be protected against infection from the air. In some regions this is a difficult problem. After prolonged anæsthesia, vomiting or coughing may lead to the entrance of air. In my own practice, violent vomiting has been an occasional cause of suppuration in the lower ends of ovariotomy wounds. After amputation of the breast, coughing, vomiting, or the movements of the arm may allow air to enter. In the root of the neck, coughing, vomiting, and movements of the head may cause the same disaster. Scrotal wounds and wounds in the groins require special precautions. Precautions are also required against infection by the soaking of fluids through the dressing from the exterior. In scrotal wounds the dressing may be wetted with urine. In wounds beneath the jaw saliva may dribble down. Moreover, blood or other fluids may soak from the wound and appear at the margin of the dressing. This constitutes a track by which bacteria speedily reach the wound. At present we are using the following dressing:—

First, the skin is dusted with finely powdered crystals of iodoform. This is sterile, and should the skin by any chance contain bacteria, they cannot grow in the iodoform. Further, I have several times seen wounds exposed to view, but protected by the layer of iodoform. Finally, it prevents dermatitis, and the itching and irritation with which that is attended. Over the iodoform a layer of 5 per cent carbolic gauze, wrung out in biniodide of mercury lotion, is placed. The carbolic gauze is chosen because it is unirritating, and can be left on until the wound is healed. It is usually removed on the eighth or tenth day. When the skin is thin or delicate, dry iodoform gauze is often applied instead of carbolic. If a gauze containing sal alembroth or sublimate, or cyanide of mercury be used, care is needed to remove the great part of the mercuric salt with weak carbolic lotion. After the layer of carbolic gauze, a third layer of alembroth wool (blue wool) is applied. This wool contains 2 per cent of sal alembroth. Any blood or serum absorbed by it becomes strongly antiseptic, and bacteria from the exterior would penetrate with difficulty. It would also be some protection against fluids from the exterior. Care must be taken lest the alembroth wool touches the skin. The alembroth wool is covered over with a very large Listerian outside dressing. This consists of eight folds of carbolic gauze, together with an outside layer of waterproof jaconet. I have all outside dressings carefully cut to pattern, and provided with buckles and straps. There is much scope for skill or ingenuity in the making of outside dressings. In some places they can be adjusted so well with straps and buckles that no bandages are needed, as, for example, the scrotum. Finally, the outside dressing is securely bandaged in place. In applying the bandage the operator should endeavour to provide for equable pressure, and guard against the entrance of air.

Subsequent Treatment.—Operations of any magnitude and duration are followed by *shock*. If this be excessive, special treatment may be needed. Warmth, brandy, strychnine, elevation of feet, auto-transfusion, rectal injections of saline solution, intravenous injections of saline solution, may have to be resorted to. The shock is followed by reaction, during which the pulse and temperature rise. Afterwards the patient has a normal pulse and temperature, and can have any light diet such as is suitable for a person lying in bed and taking no exercise. Stimulants and tobacco may be used in moderation. In past time the effects of septic infection were often attributed to food and alcohol.

Aseptic wounds are painless, but should pain be felt at the wound, or even much discomfort, or should the temperature rise or pulse accelerate, then the wound should be dressed and examined.

The Subsequent Dressings.—Drainage tubes are removed at the end of twenty-four or forty-eight hours. Any oozing of blood will have ceased by this time. When such an early dressing would be attended with pain, a longer delay may be allowed. An amputation through the thigh, or a Syme's amputation, may be left until the fifth day or longer. Should, however, the dressing require to be renewed because blood or serum may have reached its edges, then the opportunity is taken to remove the drainage tube. The operation of dressing the wound requires the greatest care, and is guided by exactly the same principles as the original operation. A freshly made wound is very susceptible to infection, and the results of infection at any of the dressings may be disastrous. Therefore the bowls, materials, and instruments used ought all to be sterilised. The hands of the surgeon or of his assistants ought also to be sterilised. Properly disinfected towels should be provided to surround the wound and make a "field of operation." An assistant or nurse should remove the bandages and outside coverings. The wound should be exposed for as short a time as possible. Usually it is instantly dusted with a layer of iodoform powder or covered with aseptic gauze, after which the drainage tube is taken out. Afterwards the same kind of dressing as that which was used at the operation is applied. The disinfection of the skin is now so perfect that it is unnecessary to apply any disinfecting lotions before removal of the stitches. Everything beneath the dressing is sterile. The stitches, therefore, cannot infect the wound when they are withdrawn. Generally speaking, the wound ought to be kept as dry as possible. If the cut edges come apart, the removal of the stitches ought to be delayed also when they hold together the abdominal walls. Aseptic sutures may remain for many weeks. It is most objectionable to place strapping under the dressing. This practice not infrequently leads to suppuration.

In some regions occlusive dressings of iodoform collodion are of great value, as in plastic operations about the face and neck, or after circumcision. This is easily provided by simply soaking iodoform gauze in flexible collodion, and applying it in several layers over the wound. At the same time some fresh iodoform is dusted on.

When the field of operation contains an ulcerated surface, as for example, an ulcerated scirrhus of the breast, a similar occlusive dressing is applied and kept on during the operation.

The Evidences of Asepticity.—The clinical evidences of asepticity are general and local. When the effects of the anæsthesia have passed away and shock has been followed by reaction, the pulse and temperature fall, the appetite returns, and the patient is like one recovering from a simple fracture. An elevation of the temperature or pulse does not usually indicate infection of the wound, but may be caused by dermatitis, bronchitis, or some inter-current malady. Pain in the back is almost constant after fractures and operations, and probably indicates hypostatic engorgement of the muscles. Pain about the attachments of the diaphragm often follows anæsthesia, especially when the breathing has been forcible and violent.

The local clinical signs of aseptic repair are *absence of pain* in the wound, and when the dressing is removed an entire absence of redness or swelling. Aseptic wounds are *quite dry*, and any moisture is almost certainly indicative of sepsis. But the only final and scientific proof of asepsis is obtained by testing the wound with culture media. For this purpose a suture is usually dropped into nutrient broth, or a gelatine tube is inoculated with a platinum wire drawn along the wound. All this is done with the usual bacteriological precautions. Of late we have frequently succeeded in obtaining ideal results. By this I mean that the skin of the patient, of the surgeon, of the assistants, the ligatures, towels, sponges, and finally the wound itself, were all submitted to bacteriological tests, and proved to be sterile.

In the year 1898, sixteen hundred and twenty-nine operations were performed in the wards and operation theatres of St. Bartholomew's Hospital (see Hospital Reports). This does not include the ophthalmic operations, or a vast number of operations done in the surgery and out-patient departments. No operation was followed by pyæmia, septicæmia, erysipelas. We owe these results to the work and teaching of Lord Lister and to the conscientious care with which the house - surgeons, sisters and nurses perform their duty. The systematic use of culture media for testing has proved a most valuable safeguard, and has done much to raise the standard of efficiency. No modern scientific surgeon now omits this precaution.

Antiseptic Surgery and Antiseptics.

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Antiseptic Surgery and Antiseptics.—*Anti*, against; *septos*, putrid. The term antiseptic is now used with more than one meaning. Originally it was revived to designate the wound treatment which aimed at the prevention of putrefaction by the use of chemicals. Ultimately, the prevention of all forms of sepsis and of suppuration was included within its scope. During the last few years, surgery, which aimed at the exclusion of bacteria by heat and sterilised fluids, and without the use of chemicals, has been by some named "aseptic," and that which relied upon chemicals for the exclusion of bacteria called "antiseptic." Inconsistencies continually arose. Chemicals had to be used for the disinfection of the hands and skin, and of septic wounds; heat had to be used for instruments and silk. A difficulty also arose in designating the mixed method, which utilises both heat and chemicals. But surgery is progressive, and, as it becomes more scientific, can be defined with greater precision. Thus, the term aseptic is applied to surgery which aims at the absolute exclusion of all bacteria. Obviously, if all are excluded, sepsis cannot occur. To attain this end the surgeon has to use various means which kill bacteria outright. That which kills bacteria is called a disinfectant, the process of killing them is called disinfection, and the result is asepsis. Agents which prevent or retard the growth and action of bacteria are called antiseptic.

The chief methods of disinfection have been referred to under the heading "aseptic surgery." Further information is given in the article "Disinfection."

Actions of Antiseptics—

Exclusion.—But it is also requisite to exclude bacteria from the wound, both during the operation and during the process of repair. For this purpose various chemicals are now in the form of lotions, powders, and dressings, and are called antiseptics. The final result is intended to be the total absence of bacteria-asepsis.

Inhibition.—Antiseptics are also used with another object in view, viz. to prevent bacteria from multiplying in or about the wound. In Koch's experiments with sublimate, the spores of anthrax were not killed but ceased to germinate. As Klein aptly puts it, the spore or bacterium is "stunned" by the chemical. This inhibitory action of antiseptics is their most valuable property.

Attenuation.—Besides limiting the number of bacteria, antiseptics have another action. The experiments of Camberland and Roux clearly proved that when anthrax bacilli were grown in the presence of minute quantities of antiseptics, such as carbolic acid or bichromate of potash, they underwent a permanent attenuation and ceased to kill. Indeed, cultures prepared in this way have been used for purposes of vaccination to produce immunity.

Neutralisation of Ptomaines.—Another of the properties of antiseptics is illustrated by iodoform. Behring has proved that it can neutralise the toxines or ptomaines produced by bacteria. He ascertained that Brieger's cadaverin caused suppuration when introduced pure into the cellular tissue of animals. A mixture of iodoform and cadaverin did not have such an effect.

Mechanical Action.—Some antiseptic powders seem to have a mechanical action. Colonies of bacteria growing upon culture media cease to spread under a layer of finely-powdered iodoform, starch, or sugar. But this only occurs when the powder is finely triturated and dry.

Deprivation of Water.—The last point illustrates the way in which some chemicals act as antiseptics by depriving bacteria of their needful supply of moisture. A plentiful supply of water is essential to the multiplication of all bacteria; certainly, of all of those which are met with in disease. It is to be noted that in this case the chemical only acts as an antiseptic, preventing the multiplication of the bacteria. When some bacteria are deprived of water they at once form spores which are endowed with extraordinary tenacity of life; anthrax, tetanus, and the bacillus septicus are examples. Fortunately they are seldom met with in surgery.

Oxygenation.—Some chemicals owe their antiseptic properties to the power which they possess of liberating oxygen. Many of the anaerobic putrefactive bacteria perish or cease to multiply in the presence of free oxygen or of ozone. Permanganate of potash or peroxide of hydrogen act in this manner. The latter may also liberate oxygen in the more active form of ozone.

Deoxidation.—Chemicals may also act by depriving bacteria of oxygen. In the bacteriological laboratory, pyrogallic acid is used for this purpose, and prevents the growth of aerobic bacteria. Antiseptics of this kind are not of surgical value.

MODE OF ACTION OF CHEMICAL AND OTHER AGENTS.—Much remains to be learnt about the action of chemicals upon bacteria. Henle and others suggest a chemical combination may occur, so that a given quantity of the chemical can only combine with a given quantity of bacteria. A sufficiency of the chemical must therefore be provided and maintained.

In the case of carbolic acid, which is volatile, resin is used to fix it in the dressings. But chemicals combine with other substances besides bacteria. Much of the efficacy of sublimate is lost because it combines with albumen to form an insoluble albuminate of mercury. This is overcome by converting the sublimate into ammonio-mercuric chloride or sal alembroth. The biniodide of mercury owes its great efficacy to the fact that it does not enter into combination with albumen.

Some chemicals, such as alcohol, act by coagulating the microprotein of the bacterium, and possibly by depriving it of water. The alcoholic series, however, do not possess any marked antiseptic properties. They are, however, the most valuable adjuncts to enable other chemicals to penetrate grease.

It is possible that some chemicals owe their antiseptic properties to decomposition, which is supposed to occur when bacteria are present. For instance, it has been stated that iodoform liberated iodine in the presence of pyogenic or putrefactive bacteria. Although I have kept mixtures of putrid pus and iodoform at body temperature for twenty-four hours, I have been unable to confirm this statement.

The antiseptic properties which have been mentioned are possessed in varying degrees by *light, cold, heat, electricity, some mechanical agencies*, and by a *vast number of chemical substances.* To this list might be added the fluids and tissues of the healthy human body, which are known to have considerable power to kill or prevent the growth of bacteria.

The strict definition of disinfectants and of antiseptics is not a mere logical quibble. Perfect safety in surgery is only obtained by the absolute exclusion of all bacteria. This is of course impossible if the disinfectants did not kill or the antiseptics exclude. In practice, however, it is usually most difficult to say where disinfection ends and antisepsis begins, or *vice versa;* or again, where disinfection and antisepsis overlap. Nevertheless, the patient's life often depends upon this kind of knowledge.

It is obvious that the antiseptics are exceedingly complex both in their mode of action and in their degree of action against bacteria. Their use requires a certain degree of skill, and the result is apt to be uncertain, unless constantly checked by the test of culture media. Thus it follows that there is room for the widest differences of opinion as to the value of various chemical substances. Before, however, a chemical is chosen, it ought to be submitted to a thorough test in the laboratory. This often proves that disinfecting properties are absent and the antiseptic are feeble. Further, its exact composition should be known, and also that it does not vary. This proviso excludes the various patent panaceas.

THE SCIENTIFIC TESTING of antiseptic substances has yielded rather astonishing results, and has shown many to possess no disinfectant powers and but feeble antiseptic. The test is usually conducted after Koch's method. Silk or cotton thread is sterilised with steam or boiling water. Pieces an inch long are soaked in a virulent culture of anthrax. This ought to have been grown at body temperature for a week, and be full of spores. After having been soaked in anthrax the bits of thread are dried in a sterilised capsule in a warm incubator. They are then soaked in the antiseptic and washed in sterilised distilled water to remove the chemical. They are then put on the surface of a culture medium or dropped into broth, or into a gelatine tube which has been melted to receive them. Finally, the culture is put into the incubator at body temperature. Koch proceeded in this way and obtained no growth. He therefore concluded the spores had been killed by the various chemicals, or their growth delayed. Also, in case the water had not removed all the antiseptic, he washed the strings in alcohol. Geppert, however, has clearly shown that Koch failed to remove sublimate by merely using water or alcohol. He found that anthrax still grew after an exposure of twelve minutes in 1 per cent sublimate solution, provided the threads were afterwards soaked in a thirty per cent solution of ammonium sulphide.

CHOICE OF ANTISEPTIC.—THE IDEAL ANTISEPTIC would have to fulfil a vast number of conditions, some of which are incompatible: (*First*) To kill bacteria and prevent their growth; (*second*) not to poison the patient; (*third*) not to injure the tissues, mucous membranes, or skin; (*fourth*) to act speedily; (*fifth*) to act continuously; (*sixth*) to penetrate; (*seventh*) not to volatilise; (*eighth*) to be soluble in water and spirit; (*ninth*) not to combine with albumen or fat; (*tenth*) not to injure instruments or garments.

The various chemicals at our disposal possess these properties in varying degrees. A part of the practice of antiseptic surgery consists in using the chemical which is most suitable for the kind of infection which has to be overcome. For instance, a solution of nitrate of silver is not very suitable as an application to a septic wound, although marvellously efficacious for a septic urinary bladder.

I have already said that the antiseptic properties of sublimate are done away with or diminished by combination with albumen. The antiseptic properties of carbolic acid are destroyed in the same way. A great many oily substances are also inimical. The experiments of Koch showed that carbolic oil possessed hardly any value as a disinfectant, and but feeble antiseptic properties. The same applies to mixtures of chemicals with glycerine, lanoline, lard, and the petroleum compounds. The greasiness of the human skin is one of the chief obstacles to its disinfection and antisepsis. The action of all soluble antiseptics is greatest when they are dissolved in a thin fluid, like water. The addition of alcohol sometimes helps by enabling the antiseptic to penetrate.

Although the disinfecting properties of chemicals are so feeble, nevertheless their antiseptic properties are sufficient for the needs of surgical work. Anthrax grows after its *spores* have soaked four hours in 1 in 1000 solution of sublimate, provided all the chemical is removed with ammonium sulphide (Kanthack). According to Koch, three parts of sublimate in a million parts of water arrests the development of anthrax *bacilli*. The ordinary pyogenic organisms are certainly not more resistant than anthrax. Therefore after instruments, sponges, ligatures, towels, hands, and so forth, have been disinfected, very dilute solutions of chemicals can be relied upon to maintain asepsis. Each surgeon has his own predilections. We are accustomed to use 1 in 60 carbolic acid lotion or 1 in 2000 to 1 in 4000 biniodide of mercury lotion.

It is exceedingly difficult to obtain an antiseptic dressing which conforms to the requirements. Those which consist of wool or gauze impregnated with mercuric compounds are exceedingly efficient antiseptics, but are apt to cause dermatitis. Those which contain boric acid, iodoform, salicylic acid, or eucalyptus are unirritating, but, at the same time, comparatively feeble antiseptics. Carbolic gauze (5 per cent) is a fairly satisfactory antiseptic, and seldom causes dermatitis.

Aspergillosis. *See* LUNGS.

Asphyxia.

(*Asphyxia neonatorum* (*apparent still-birth*)—*Resuscitation, Artificial Respiration.*)

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  Irrespirable Gases.
  Chloroform Inhalations.

Definitions.—Asphyxia, which etymologically means pulselessness, is the condition of the organism when consciousness has ceased in consequence of defect in the aeration of the blood. In the middle of the century a persevering attempt was made to replace the term, on the ground of literary accuracy, by apnœa (respirationlessness). Apnœa is now, however, the name definitely assigned to the condition in which the respiratory movements are suspended in consequence of over-aeration of the blood. The two terms are thus assigned to contrasted conditions. For the intermediate conditions a graduated series of terms is used. Eupnœa is the condition of easy normal respiration; hyperpnœa, deepened respiration, such as follows on exertion; dyspnœa, respiration in which the accessory muscles are called into play; orthopnœa is that variety of dyspnœa in which the respiratory distress is increased in the horizontal position.

The Phenomena of Asphyxia may be produced rapidly, *e.g.* by sudden obstruction of the trachea, or may develop gradually. When the trachea is suddenly occluded, there is (1) a period of quiet of from twenty to thirty seconds, longer or shorter according as the obstruction was made at the end of inspiration or expiration. Respiratory movements of both inspiration and expiration then follow, and rapidly increase in depth and rate, until about a minute after occlusion there are powerful convulsions, expiratory movements predominating. Exhaustion gradually sets in, and the respiratory movements change to a more inspiratory type, and become slower, longer, and irregular, until they gradually cease. (2) The arterial blood-pressure begins to rise immediately on occlusion and reaches a point far above normal. From this it quickly falls until below normal, and then more slowly it approaches zero. Till the early part of the fall of blood-pressure the heart-beats are forcible and slow, but afterwards become more rapid and feeble, and at length, some time after the stoppage of respiratory movements, cease. The venous pressure also rises enormously as the result of the slowing of the heart, and this rise is increased by the convulsive contractions of the muscles. The right heart dilates under this pressure, and ultimately, from this and the weakening of the heart-movements and the malnutrition of the heart-wall from the venosity of the blood supplied to it, becomes engorged. (3) Fully a minute after the occlusion, consciousness is lost, the sphincters are relaxed and urine and fæces are usually voided. The reflexes are abolished in succession and muscular tone is lost.

In the production of the phenomena consequent on defective aeration of the blood, two factors are operative, a deficiency of oxygen and an excess of carbonic acid. The controversy as to their relative importance is not entirely closed, but it seems clear that each can produce the hyperpnœa, the rise of blood-pressure and the unconsciousness, with the cessation of respiratory and later of circulatory movements, which ends in death by asphyxia. Carbon dioxide excess is operative even when there is so much oxygen present that the blood retains its arterial colour, and simple decrease of oxygen below half the normal proportion inaugurates a similar series of phenomena. At the close of an asphyxia ending in death the blood is entirely deoxygenated. It seems thus certain that in actual practice both factors co-operate.

After death by asphyxia, apart from the engorgement of the right side of the heart and the systemic veins, even after the left side of the heart has been emptied by rigor mortis, none of the appearances can be said to be either uniform or characteristic. The whole pulmonary circulation is gorged and the mucous membrane of trachea and bronchi injected.

Asphyxia livida and *Asphyxia pallida.*—In the process of asphyxia, there is a stage when the patient has a livid appearance, the muscular tone is preserved, the blood-pressure is high, and the heart-beats, though slow, are powerful; this is succeeded by a condition in which the peripheral vessels are empty and the skin pale, the muscular tone lost and the heart-beats feeble. The prospect for the patient is so different, according as he has reached the first or the second of these stages, that they have long been clinically distinguished by the names livid and pallid, especially in the asphyxia of new-born children.

Causes of Asphyxia.—There are conditions in which air is replaced by other gases, *e.g.* carbon dioxide, as we have already seen, and carbon monoxide. Carbon monoxide produces a more stable compound than oxyhæmoglobin, and leads to complete oxygen starvation. It is the main agent in death after colliery explosions, and in poisoning by lighting gas. 2. Conditions in which air is excluded mechanically from the lungs. Such are, the spasm of the glottis caused by irrespirable gases, pure carbon dioxide being supposed to act in this way; obstruction by impaction of food or foreign bodies in the pharynx; suffocation and overlaying; strangulation and hanging; drowning; some diseased conditions, such as, œdema glottidis, diphtheria, œdema of the lungs and catarrhal pneumonia; tetanic contraction of the respiratory muscles, as in tetanus and strychnine poisoning. 3. Conditions in which the respiratory movements stop from paralysis of the central nervous mechanism of respiration, as in poisoning by opium or chloroform. During birth, prolonged pressure on the head may produce asphyxia in this way. 4. During birth, pressure on the umbilical cord may interrupt the flow of aerated blood from the placenta and produce asphyxia. This sometimes gives rise to respiratory movements, and if these take place before air can reach the nose we may have asphyxia during birth, produced as in the second class of conditions.

The rapidity of asphyxia and conditions which affect it.—It is not easy to fix the limit beyond which asphyxia must inevitably issue in death, but usually when air has been shut off from the lungs for five minutes, the heart does not recover. After exertion, when the respiratory need of the tissues is greater, the asphyxia runs a shorter course. In children, at birth the rate of physiological exchange is low, and they recover after much longer periods of asphyxia than older animals. During syncope the oxygen need is low, and this favours recovery after longer periods than usual. Low temperature down to about 60° F. also prolongs the period of asphyxia. Thus in drowning the period of submersion with possible recovery is much greater at the lower temperatures, as was shown by the old but well-established experiments of Edwards.

Resuscitation.—If at any point in the process of asphyxia the cause producing it be removed, the process may be reversed and recovery take place. By properly adapted measures we may aid this. In most cases, apart from asphyxia produced by poisoning or disease, we can have no access to the resources of a hospital, and must deal with the emergency with means always at hand. For this reason the administration of oxygen and the electric stimulation of the respiratory muscles need not be considered here. The exact *indications for treatment* will in each case depend on the stage which the asphyxia has reached. It is essential that a supply of aerated blood should be circulated through the nervous centres till they are sufficiently active to carry on automatically the circulation and the respiration. This may at times be aided by the inversion of the patient. If the respiratory centre is not yet paralysed it may react to external stimuli, such as dashing cold water on the skin, tickling the larynx or traction on the tongue. In this case we probably only hasten the recovery, which would occur spontaneously. If the respiratory centre is anæsthetic, respiratory movements are suspended, and we must replace them by such manœuvres as produce entrance and exit of air into the lungs. If the asphyxia is still more advanced and the circulation almost stopped, we must see that the manœuvres we adopt are such as not only promote the access of air to the lungs, but force the blood from the gorged heart, so that it passes on through the lungs and through the nerve centres. The essential need in these two latter classes of conditions is to supply for the time being an artificial respiration and an artificial circulation.

Historical.—The earliest method of supplying air to the lungs in asphyxia was that of inflation. This was performed at first from mouth to mouth, the patient's nostrils being held; later, specially devised laryngeal catheters were used, and eventually the inflating mouth was replaced by special bellows, which had the advantage of supplying air with more oxygen and less carbon dioxide. In addition to the inflation it was usual to employ manual compression of the chest to assist the expiration. The idea of imitating the respiratory movements is due to Leroy, who devised a method of compressing the chest by traction on the crossed ends of a many-tailed bandage passed under it, alternately with relaxation of the pressure to secure inspiration. After this no step of importance was taken till the second half of this century, but since that we have had a rapid succession of methods, which, even with the authors' names attached, is somewhat bewildering until we realise that they are mostly rearrangements or rediscoveries of a few fundamental manœuvres whose principles were revealed to the earlier writers. In 1856 Marshall Hall published his "postural" treatment in drowning, and Silvester had already discovered his "physiological" method of raising the chest walls by pulling on the arm muscles, and in 1859 Schultze was using his method of "swinging" apparently still-born children, though he did not publish it for several years. Since that time many modifications or combinations of the elements of these methods have been published as new, but whether they are improvements or not, these new methods, with the exceptions of Schüller's mode of grasping the edge of the ribs and Laborde's method of rhythmic traction on the tongue, contain nothing that is not contained in the earlier methods.

In *proof of the value of each method* of artificial respiration, clinical evidence rapidly accumulates, but for several reasons this is almost valueless, and leads to conclusions which are opinionative merely and not scientific. It is impossible to say beforehand that any given case of asphyxia will not recover spontaneously. Most cases of livid asphyxia, and some of pallid, recover without the least aid from the physician. As a rule, too, the physician is not in an entirely judicial frame of mind in dealing with a case of asphyxia, and if he is, the excitement of the bystanders is enough to disturb it; he often fails to note the stage of the asphyxia—at least he usually fails to record it. In many cases the recovery is a long, slow process, during which the patience of the operator is not proof against the temptation to try another method, and at the end of any series of such changes, the method last tried before recovery is the efficient one. We are therefore indebted to the methods of the physiological laboratory for a trustworthy criterion as to the relative efficiency of various manœuvres for introducing air into the chest. The question has not been so often investigated on adults as on children, but the reports of the Royal Medico-Chirurgical Society on Suspended Animation, and that of Brosch, furnish much useful information, while the careful and extensive investigations of Champneys will serve as a type of the work that has been done on infants. In the matter of still-born children it is to be remembered that we start with the lungs in a completely airless condition, so that the problem is not entirely the same for them as it is for the adult. While the general opinion is that artificial respiration is useful in children, we must modestly remember that Ahlfeld says, "All apparently still-born children, who are otherwise capable of surviving, need, besides the clearing of the air-passages, merely to be kept in warm water to come bit by bit to free breathing. Skin stimuli shorten the period of incomplete respiration."

The manœuvres of artificial respiration may be most easily understood when tabulated as follows:—

Inspiratory—*A.* Inflation. 1. Mouth to mouth. 2. By catheter.
B. Increasing the thoracic dimensions.

1. Raising the anterior wall.
    - (*a*) By pulling up the rib margin (Schüller).
    - (*b*) By pulling on the pectoral muscles (Silvester, Forest).
    - (*c*) By pulling on all the shoulder muscles (Bain, Pacini, Brosch).
    - (*d*) By the weight of the arms (Byrd).
2. Depression of the diaphragm, by throwing down the abdominal contents.
    - (*a*) By their weight (Byrd, Dew, Schultze).
    - (*b*) By movement (swinging) (Schultze).
3. Elastic recoil of thoracic wall after compression (Hall, Howard, Duke, Prochownik).

Expiratory—

1. Compression of the thorax.[1]
    - (*a*) By the weight of the trunk (Hall).
    - (*b*) By direct manual pressure (Howard, Duke, Prochownik, Schüller).
    - (*c*) By indirect pressure through the patient's arms (Silvester, Brosch).
    - (*d*) By resisting the trunk in movement (Schultze).
    - (*e*) By flexion of the trunk (Byrd, Schrœder, Dew, Schultze, Forest).
    - (*f*) By bandage passed round the chest (Leroy, Flashar).
2. Elastic recoil of the thorax after distension (Bain, Pacini, Silvester).

It is evident that manœuvres involving flexion or swinging of the trunk are adapted to the child rather than the adult. The experimental examination of these methods has shown that the elastic recoil of the thorax after compression is not a very efficient method of securing inspiration, and that the largest air introduction is secured by the modifications of the Silvester method. For the performance of this and of the other most useful methods, the detailed instructions are given in the following paragraph.

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[1] Brosch has shown that there is a risk of the contents of a full stomach, expressed during the expiratory movement, being sucked into the trachea in the next inspiration. For this reason great pressure on the epigastrium should be avoided, and the head kept well back.

The methods of artificial respiration.—It is assumed that, preliminary to any method, the air-passages have been freed from obstruction.

I. Inflation—(1) Mouth to mouth. The patient's nostrils being held, a handkerchief is laid over the mouth, to which the operator applies his own mouth and blows until the lungs expand. The air is then expelled by pressure on the front of the chest, and the whole manœuvre is repeated regularly. Some pressure should be made on the epigastrium to prevent the air entering the stomach instead of the lungs. (2) By catheter. In infants, the flexible catheter, which the accoucheur has always sterile in his bag, can be readily introduced into the larynx under the guidance of the finger; the operator then alternately blows through it and allows the air thus introduced to escape. *Notes.*—Inflation must always be performed with extreme care and gentleness, for, as Leroy showed at the beginning of the century, it is liable to produce emphysema and rupture of the lung, and so to confirm the fatal issue which we would avoid. It has the additional drawback that authentic records exist of the production of a series of cases of tuberculosis, where the inflation was performed by a phthisical midwife.

II. Silvester's method (modified). The patient being in the dorsal position, the back of the thorax is raised about a foot from the ground or table by a pad, which may be the operator's coat or anything else at hand, but must be firm. The tongue is drawn forward and fixed with a band passed round the chin. The operator grasps the arms above the elbows, and moves them upwards and outwards till they are at the sides of the head, then they are forcibly depressed. This completes the inspiration. For the expiration the arms are placed together on the front of the chest, and pressed as strongly as possible directly backwards. The manœuvre is repeated regularly. *Notes.*—These are practically the directions of Brosch, and embody the most effective modes of carrying out the Silvester method, which raises the chest-wall by pulling on the arms. When assistance can be obtained, it is advantageous to have the feet held. Silvester methods are somewhat exhausting for the operator, and it is well when two can relieve each other if the respiration has to be maintained for a considerable time. The experimental tests show that the amount of air thus artificially introduced is greater than that in ordinary respiration.

III. Schüller's method. The fingers are pushed under the margin of the ribs, which are then alternately pulled up and depressed. *Notes.*—This method is less exhausting for the operator, and is specially useful where prolonged rather than energetic aeration is needed, *e.g.* in narcotic poisoning.

IV. Schultze's method for children. The child being grasped so that the operator's thumbs rest loosely on the front of its chest, his forefingers enter the axillæ from behind and his fingers are spread over its back, while the head is supported by the hollows of his hands, he, standing a little astraddle and bending a little forward, holds it at arm's length hanging down. Without delay he, swinging his arms upwards straight till they have passed the horizontal, flexes the elbows a little, and the momentum carries the child's pelvis and legs gently on and so compresses its abdomen, while the whole weight of its body presses the thorax against the operator's thumbs. The child is then swung downward again, and the pressure against chest-wall and diaphragm relieved. It is advantageous to wrap the child loosely in a dry cloth. *Notes.*—Much discussion has taken place as to whether this method is apt to produce serious injury to the child, and making all allowance for errors of observation it is impossible to give an entirely favourable verdict. Ahlfeld had a sudden death in a child on which he was merely demonstrating the method. That it can aerate the lungs in the entire absence of spontaneous respiration is clear from the post-mortem examination of children who have died from causes other than asphyxia,—*e.g.*, cerebral hæmorrhage.

V. Buist's method for children. The child is laid face downwards on the operator's right hand, with the head away from him, the upper and lower parts of the body being dependent over the edges of the hand. The child is then rolled over and transferred with a slight throw to the left hand, when, the arms and legs being again dependent, the arms pull up the thorax wall and the abdominal contents move down and pull down the diaphragm. The positions are then regularly alternated. *Notes.*—This method was devised to meet the exigencies of the small space available in some cases in domestic practice, and while less violent than Schultze's method embodies its special feature, descent of the diaphragm in the inspiratory movement. Manual compression can be exerted by the fingers of the left hand while the child is in the expiratory position, and if desired this may be specially directed to the cardiac region.

The Circulation during Artificial Respiration.—In deep asphyxia the arterial blood pressure is low, and the pulmonary circulation gorged. During the expiratory movements the increase of the pressure within the chest affects the heart and vessels as well as the lungs, and tends to force on some of the blood into the arteries. During the inspiratory movement reflux is prevented by the valves, and in this way we get an onward movement of blood, while the emptying of the heart cavities and the alternate filling relieves their paralytic distension. All methods of artificial respiration thus provide to a certain extent for artificial circulation as well, but it is possible to help this by pressure specially directed to the heart. This may be by compressing it by a hand pushed under the ribs at the epigastrium, or by using, in the interval of the slower movements of respiration, more rapid rhythmic pressure on the præcordia.

Adjuvants to artificial respiration in resuscitation.—(1) In almost all the directions given for the resuscitation of persons from drowning, and of apparently still-born children, the use of the warm bath is recommended. This practice is, nevertheless, so much against the available scientific evidence, that it is questionable whether it deserves its empirical repute. The experiments of Edwards, Brown-Sequard, and Marshall Hall show that more prolonged asphyxia can be survived at lower temperatures down to about 60° F. The temperature of the animal does not of course sink to that of the surrounding medium, and the experiments seem to show that the depression which occurs is advantageous. While, therefore, the advantage of the warm bath when the respiration has been established is undoubted, our first efforts in resuscitation must be directed to the artificial respiration. The argument is not of cŏurse to be extended to exclude the prevention of further heat loss. (2) Other adjuvants at our disposal are the various methods of applying stimuli to the skin or mucous membranes, such as the slapping, in children, the rubbing of the face with a dry towel, the tickling of the pharynx or nostrils, or the use of ammonia or other volatile agents. All of these methods depend on the reflex activity of the nervous system, and though they may shorten the period of incomplete respiration they can hardly be relied upon when the organic reflexes are so deeply sunken as to call for artificial imitation. (3) When the heart is flagging or almost stopped, a pad or sponge dipped in boiling water should be applied to the præcordia. Whether the heat is able, through the chest-wall, to stimulate the cardiac muscle directly, or whatever mode the action may take, there is no doubt as to the efficiency of the method, which is specially useful in some cases of chloroform anæsthesia. The difficulty of explaining the scald is less than that of reconciling the relatives to a death under the anæsthetic for a, perhaps, trivial operation.

The question, *How long are efforts at resuscitation to be continued?* does not admit of a simple answer. Unless the heart is beating, however feebly, it is probably useless to make any effort unless in the new-born child, when it may be worth while to try the effect of rhythmic compression of the heart region. In other cases, unless the heart-beats improve in force and number, it is improbable that our efforts will be successful, and though there are records of cases in which heroic perseverance for hours have been rewarded with the recovery of the patient, an hour may be looked upon as the limit of useful effort, except in cases of poisoning, when the resources of a well-trained police force or ambulance brigade may be requisitioned to maintain the treatment for a much longer period. In some cases, which occur in hospital, in which the heart's action is well sustained though there seems little tendency to spontaneous respiration, it might be well to do tracheotomy, and apply the respiration apparatus of the laboratory, with which the artificial respiration might be maintained for much longer periods. There is no experience more distressing than a failure to continue artificial respiration while the heart still beats. In new-born children, which do not quickly react, it is worth while to reconsider the circumstances of the delivery with a view to the diagnosis of a probable cerebral injury.

DIRECTIONS FOR SPECIAL CONDITIONS.—*New-born children.*—Clear the pharynx of slime before beginning any method. If any one can hold the feet for you, Silvester's method is a good one, otherwise use Buist's method or Schultze's. Alternate plunging in hot and cold water is an excellent stimulus, but each should be momentary. Schultze's method cannot be applied until the umbilical cord has been divided. (In the middle of last century it was a common practice to lay the placenta on the child's belly, and last year Stringer recommends the exposure of the placenta to the air as the rational treatment of asphyxia. Like every other he is able to adduce plenty of clinical evidence of success.)

Drowning.—As in this condition the trachea often contains frothy mucus, it is well to begin with a forcible expiratory movement. Then go on with Silvester's method. It adds to the effect of this if the feet are held.

In asphyxia due to irrespirable gases we need to use the most vigorous means at our disposal for the introduction of air. This is especially the case with carbon monoxide (after-damp), which replaces the oxygen in its combination with hæmoglobin.

Chloroform inhalation is apt at several stages to be complicated with cessation of respiration. The special treatment to be adopted is described under "Anæsthetics."

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Aspirator, Uses of.

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1. *Diagnostic.*
2. *Therapeutic.*

The method of exploring fluid-containing cavities, known as aspiration, was introduced into surgery by Dieulafoy in 1869. The principle is to bring the contents of the cavity into communication with a "previously formed vacuum," through the medium of a hollow needle, in order that the negative pressure of the vacuum may effect the withdrawal of the fluid.

Apparatus.—Of the many forms of aspirator which have been devised, those of Dieulafoy and Potain are the most generally acceptable. *Dieulafoy's instrument* consists of a strong glass syringe, fitted at the nozzle with a two-way tap. One passage connects the cylinder, by means of a rubber tube, with a terminal hollow needle fitted with a stop-cock. The other, also bearing a stop-cock, carries the fluid aspirated away from the cylinder. If both stop-cocks be closed and the piston withdrawn, a vacuum is formed in the syringe, into which the fluid is sucked when the needle has been introduced into the cavity to be tapped, and its stop-cock opened. The stop-cock of the needle-tube is now closed and that of the outlet-tube opened, so that the fluid may escape into a receptacle. This process is repeated till the cavity is emptied. This instrument may be so constructed that the exhausting syringe merely starts a syphon action, by means of which the fluid is evacuated.

In *Potain's aspirator*, by means of an exhausting syringe, a vacuum is formed in a glass bottle, which is brought into communication with the fluid to be withdrawn through a trochar and cannula attached to a flexible rubber tube. By an arrangement of stop-cocks the trochar can be withdrawn without the admission of air, and the vacuum permitted to act on the fluid. Should the bottle become filled, or the pressure in it cease to be negative, by the use of the appropriate stop-cocks, it may be removed, emptied, and re-exhausted without withdrawing the cannula or admitting air into the cavity. Potain's instrument, although a little complicated, is perhaps the best aspirator for general use.

Technique.—Whichever apparatus is employed, it is essential that absolute asepsis of every part of it be ensured. Before introducing the needle, the skin of the part is purified by washing with soap and water, turpentine or lysol (1 in 100), and carbolic lotion (1 in 20). The apparatus is tested to see that it is in working order by running a stream of antiseptic fluid through it. After re-establishing the vacuum, the needle is lubricated with an antiseptic oil, and smartly passed through the skin. In using Dieulafoy's apparatus, so soon as the needle-point is buried in the tissues it is connected with the vacuum by turning the appropriate stop-cock, in order that fluid may flow whenever it is reached, and to avoid the error of passing the needle right through a collection of fluid without tapping it. With Potain's instrument the trochar must be in the fluid before the vacuum is opened. Should the flow suddenly cease, a flake of lymph or thick pus has probably blocked the needle. This may be dislodged by the use of a blunt stylet, or by reinjecting some of the fluid, according to the structure of the apparatus in use. Aspiration must be stopped at once should blood flow in any quantity, or should the patient show signs of collapse, or of irritative cough. In withdrawing the needle there is a risk of infecting its track by pus escaping from the point. This may to some extent be obviated by keeping the vacuum in action during the withdrawal. The puncture is bathed with an antiseptic lotion, and sealed with collodion. Should the needle meet with much resistance on its way in, it must be withdrawn and reintroduced at a different point. Any attempt to guide it past an obstruction by twisting is very apt to break it across.

After use, the whole apparatus must be thoroughly cleaned with antiseptics, dried, and carefully put away. Needles should be washed through with hot water, then dried in a spirit flame, which prevents rusting. Avoid strong carbolic lotion for the purification of the needles both before and after use, as it coagulates albuminous fluids in the lumen, and leads to blockage. It is an advantage to pass a silver wire stylet into the lumen of the needle before putting it aside, to prevent occlusion by rust.

APPLICATIONS.—It must be said that the advent of the antiseptic principle into surgery has largely done away with the necessity for aspiration in many circumstances where it would otherwise have played a most important *rôle*. There are few conditions now where an aseptic incision is not better surgery than the blind plunge of an aspirating needle.

The indications for the use of the aspirator naturally fall into two classes—(1) for diagnostic purposes; and (2) for therapeutic purposes.

(1) *For Diagnostic purposes.*—Positive findings have great value, but negative findings almost none, especially when a short fine hypodermic needle is employed. The fluid-containing cavity may be missed, the fluid may be too thick to pass through the needle, or no cavity may exist.

Exploratory aspiration perhaps finds its most useful fields in detecting the presence of fluid in certain situations where physical signs are either absent or conflicting, *e.g.* in the pleural cavity of children; in the diagnosis between serous and purulent collections in the pleural cavity; in doubtful effusions into joints; and in distinguishing between abscesses, cysts, bursal, and other fluid swellings in various situations. When the needle has to traverse a presumably healthy serous space before reaching the part to be explored, the process becomes a dangerous one, and had better give place to an exploratory incision. It is, therefore, not available for determining between hydatid cysts and abscess of the liver; for deciding the character of fluid in the gall-bladder; for investigating pancreatic, mesenteric, ovarian, or other intra-peritoneal cysts. It is still more dangerous to introduce an aspirator needle into the lumen of the intestine. In all these conditions the risk of sudden rupture, or of slow leakage of septic material into the peritoneal cavity, precludes this method of investigation.

With a healthy pleura, also, the danger of exploring tuberculous gangrenous, hydatid, or other cavities in the lung is too great to warrant its use.

The same comment applies to its employment in the diagnosis of intra-cerebral collections, and of renal swellings such as hydro-nephrosis and pyo-nephrosis.

(2) *For Therapeutic purposes.*—The objections urged against aspiration as a diagnostic measure hold with equal force against its therapeutic use.

There are, however, certain conditions where it is a valuable measure. Perhaps the commonest indication for its use is in cases of simple inflammatory *effusion into the pleural cavity*, where marked benefit usually follows one or more aspirations. Also in cases where, from cardiac insufficiency or other cause, there is an excessive transudation into serous cavities, *e.g.* the peritoneum or pleura, this procedure finds a wide field of usefulness.

Where *pericardial effusion* is interfering with the heart's action, a fine needle passed upwards and inwards through the fourth or fifth interspace, about one inch from the left side of the sternum, avoiding the internal mammary artery, which lies behind the costal cartilages, may give relief. In *pneumothorax* the withdrawal of air may favour the re-expansion of the lung; and in *hæmothorax* from injury or malignant tumour, the withdrawal of blood may remove the mechanical embarrassment to the breathing.

In cases of excessive effusion of *inflammatory fluids* or blood into the cavities of large *joints*, the removal of even a small quantity often greatly hastens the absorption of the rest, and relieves pain. Firm elastic pressure is applied after aspirating. Large *hæmatomata* in other situations are often more speedily absorbed after a quantity of the more fluid portion has been withdrawn by aspiration.

Over-distension of the bladder, from impassable stricture or enlarged prostate, may be relieved temporarily by aspirating in the middle line close above the pubis. The fluid should be drawn off slowly, and the bladder should not be completely emptied, otherwise bleeding is apt to take place from the veins of the bladder-wall suddenly deprived of the support of the urine. This procedure is not devoid of risk from leakage of urine along the needle track, especially in enlarged, thickened, and fibrous bladders. It should not be repeated more than a few times.

Abscesses in hæmophylic persons, or the large hæmatomata which often occur in such patients, and are so slow to disappear, are better treated by aspiration than by the knife.

In the course of certain operations, such as those for abscess of the liver, bronchiectatic cavities in the lungs, cerebral abscess, etc., after the organ has been exposed by incision, the aspirator is often a valuable aid in determining the exact situation of the cavity sought for.

In another group of cases aspiration is often permissible, although not always advisable. In *empyæmata*, for example, although it sometimes is followed by cure, especially in children and in pyæmic cases, success cannot be depended upon, and is more likely to follow incision and drainage. This is also true of pyæmic collections in joints and subcutaneous tissue, as well as in cases of cold abscess in connection with tuberculous disease in bone or lymphatic glands.

In chronic hydrocephalus and spina-bifida the value of aspiration is more than doubtful. In all abdominal conditions, such as hydatids or abscess of the liver, suppuration in the gall-bladder, pancreatic, ovarian, and other cysts, mesenteric or pelvic abscess, gaseous distension of the intestine, as well as in hydro- and pyo-nephrosis, and in acute abscesses generally, it is inappropriate.

Asthenopia.

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Asthenopia literally means weak vision (from ἀ-, σθένος = strength, and ὤφ). The weakness, however, which is designated by the term asthenopia is not one of defective vision (*vide* Amblyopia) but a want of the power to sustain an effort of vision.

A person who is asthenopic is unable to get the full use of his eyes, because any attempt at doing so is accompanied by more or less marked symptoms of intolerance.

This limited power of sustaining an effort to see is almost exclusively noticeable in connection with the use of the eyes for near objects. It therefore, for instance, shows itself when attempts are made to read or sew.

The complaints made by asthenopes vary very much in their nature. This is partly due in any particular case to the cause of the asthenopia. It is also due to the different way in which different individuals feel and describe their symptoms. Some observe a tired feeling in their eyes which after some time, without amounting to actual pain, becomes more or less intolerable. In others, again, reading or other near work causes pain. This pain may be referred to the eyes themselves or to the region over the eyes. Sometimes it amounts to headache.

Sometimes what is noticed is an indistinctness of objects looked at, which comes on after some little time — sooner or later according to circumstances. In reading, for instance, the letters appear to run together; a conscious effort is necessary in order to focus sufficiently sharply. Another complaint is of everything becoming black before the eyes. Pricking, burning, or itching sensations in the eyes are also frequently described.

Occasionally the train of symptoms which characterise asthenopia may be so aggravated after a time as to give rise to more general disturbance, nausea, faintness, etc.

THE CAUSES of asthenopia are various. They may be grouped under three heads:—(1) Refractive, (2) Muscular, and (3) Neurasthenic.

1. REFRACTIVE ASTHENOPIA.—All anomalies of refraction may be the chief or sole cause of the various symptoms indicative of a more or less rapid tiring from reading or other near work. The state of refraction which is, however, most apt to be associated with such symptoms, is hypermetropia. A hypermetrope is obliged to exert a greater effort of accommodation in order to focus for the reading distance than an emmetrope or myope. But this greater effort is easily made where the amplitude of accommodation is considerable as compared with the degree of hypermetropia. Such an increased effort could not well cause asthenopia in young individuals with a low or moderate degree of hypermetropia. Yet the very frequent asthenopia of young hypermetropes is often ascribed to this cause, notwithstanding that Donders long ago gave the true explanation of the association which exists between hypermetropia and *accommodative asthenopia*. It depends upon what Donders called the *relative* as compared with the *absolute* amplitude of accommodation. The relative amplitude of accommodation is the measure of the total change which can possibly be made in the accommodative effort, whilst binocular fixation is maintained for a certain definite distance. For every distance of binocular fixation there is a corresponding degree of relative amplitude of accommodation. It is possible in every case to increase the accommodation so as to focus for a nearer point than that fixed binocularly, and also to relax the accommodation so as to focus for a point further off. The relative amplitude of accommodation may then be looked upon as composed of two parts—the *positive* and the *negative*.

In the ideal condition of refraction, or emmetropia, the positive portion of the relative amplitude is much greater than the negative for binocular fixation at the ordinary reading distance. But if the emmetrope should try to read very close he soon finds that he gets tired: his eyes "give out." He is then working under adverse conditions: not on account of any difficulty there may be to maintain convergence, but owing to the positive element of the relative amplitude of accommodation being then too small to admit of a sustained effort. In other words, he becomes, in fact, under the circumstances, asthenopic. Even attempts made, after this tiring, to read at a distance which can usually be done without any conscious effort, prove more or less difficult. Before they can be renewed with comfort the eyes must be rested. What in the case of emmetropia applies only to unnecessarily near work is actually, or more or less approximately, according to the degree of hypermetropia, applicable to work done at the ordinary reading distance (11″–14″) in that state of refraction. For the reading distance the hypermetrope has a smaller surplus of positive relative accommodation, *i.e.* less reserve to fall back upon, and therefore more readily tires. In a good light and with large print, or other objects capable of forming sufficiently large retinal images when held at some distance from the eyes, no undue effort may be experienced. Under less favourable conditions, when, for instance, small print has to be read and the illumination is bad, a hypermetrope, of even a low degree, may be troubled with symptoms of asthenopia.

The symptoms most characteristic of this, the accommodative form of asthenopia, are a dull, tired, sometimes aching sensation in the forehead over the eyes and the "running together" of print when reading. But any of the other sensations already referred to may be complained of. The symptoms come on sooner or later according to the degree of the hypermetropia, the conditions under which the work is done, and the age and health of the individual.

The first tendency, whenever the effort of accommodation causes a difficulty, is to remove the book or other object further from the eyes. For a time this suffices. But the overstraining having once been necessitated often causes a similar difficulty to assert itself, under the new conditions, after the lapse of some time.

All these symptoms disappear very soon after correction (*vide* Refraction) of the hypermetropia.

Another form of refractive asthenopia is that connected with astigmatism. The symptoms characterising asthenopia from this cause are pain referred to the eyes or forehead, and often a hot or pricking sensation in the eyes. There is usually at the same time some congestion of the ocular conjunctiva, though rarely to any marked extent. The lower degrees of astigmatism more frequently give rise to this form of asthenopia than do the higher. It is more common, too, in cases in which the cornea has its greatest curvature in the horizontal or nearly horizontal meridian. The cause of the asthenopia appears to be pressure of the lids on the cornea. By such pressure an unconscious effort is made to overcome the astigmatism. The symptoms pass off on correction of the astigmatism.

Asthenopia due to myopia is rare. For most myopes little difficulty is experienced in reading or other close application of the eyes. In the higher degrees of myopia, however, at all events as long as binocular fixation is persisted in, the excessive convergence which is necessary for distant vision causes a more or less rapid tiring. This is especially the case when the myopia is active and progressive. The pain then experienced, in addition to the tiring, is referred to the back of the eyes. The treatment of such cases is that for progressive myopia (*q.v.*).

2. MUSCULAR ASTHENOPIA.—To some extent the tiring of the eyes, which results from their close application when very myopic, may be looked upon as a muscular asthenopia. Yet this kind of tiring is certainly not analogous to that which is found in the skeletal muscles after unwonted exercise. It is probably more an innervation fatigue, as discomfort is not

afterwards felt when the internal recti are associated in lateral movements with the externi.

Altogether it is doubtful whether the term muscular asthenopia, which was at one time more frequently employed than it now generally is, has any real applicability. When attention is directed to the eyes on account of symptoms of asthenopia, it is not uncommon to find latent deviations which are then often supposed to be the cause of the asthenopia. Yet similar deviations—more particularly latent convergence and divergence—are very often met with in individuals who do not complain of asthenopia at all. Latent vertical deviations (what has been called hyperphoria) do, however, seem to give rise to symptoms of fatigue, which may be removed by wearing suitable prismatic correction. (On this subject see articles on Ocular muscles and Strabismus.)

3. NEURASTHENIC ASTHENOPIA.—A very large group of cases of asthenopia are mainly *nervous* in their origin. They depend upon conditions of general nutrition or of the nervous system, which may render the most ordinary use of the eyes more or less painful or troublesome. Hysteria, neurasthenia, malnutrition, the state of convalescence after serious illness, gout, etc., are some such conditions generally associated with more or less marked asthenopia. Often there is a combination of the neurasthenic form of asthenopia with the refractive or muscular varieties. It may thus be a matter of some difficulty to determine how much of the symptoms is due to the one cause and how much to the other. The possible neurasthenic element must, in fact, be taken into consideration in the treatment of all asthenopias.

LITERATURE.—DONDERS. *Anomalies of Refraction and Accommodation.*

Asthma.

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ASTHMA (ἄω, to blow; ἆσθμα, a short-drawn breath, panting) is a term originally applied to all forms of dyspnœa, which has in course of time become more limited in its connotation. In the seventeenth and eighteenth centuries it was used for very diverse diseases in which difficulty of breathing occurs; pneumo-thorax, for example, was sometimes known as asthma aerium, whilst asthma arthriticum was one of the names of angina pectoris, but it was chiefly employed to indicate ailments characterised by recurring dyspnœa. To designate the severer paroxysmal forms of dyspnœa, the terms spasmodic or convulsive were added to the word asthma.

The introduction of more exact methods of diagnosis has led to the differentiation of some forms of paroxysmal dyspnœa from the symptom complex now known as asthma. The word asthma is indeed still used to signify dyspnœa, more or less recurrent, due to other ailments, but it is then combined with some qualifying term. Cardiac, renal or uræmic, and hæmic asthma, for example, are terms sometimes employed to indicate recurrent breathlessness in certain affections of the heart, kidneys, or blood, whilst by thymic asthma is meant an affection characterised by recurrent dyspnœa immediately due to laryngeal spasm.

But the use of the word asthma by itself is limited to that ailment which is characterised by recurrent attacks of dyspnœa connected with changes in the bronchi. The term bronchial asthma is by many regarded as synonymous with asthma, but by others a distinction is made, bronchial asthma being looked upon as a bronchitis with recurrent dyspnœa, whilst asthma is considered to be a special affection of the bronchi of which changes in the mucous membrane are a complication.

It is not possible, however, to draw clinically any distinct line between these two forms. This is well seen in so-called hay asthma, where the same cause may lead to bronchitis with slight recurring dyspnœa or to the most typical paroxysmal attacks.

ORIGIN AND COURSE.—Asthma most commonly develops out of what appears to be an ordinary catarrh. On this may supervene a sudden paroxysm of dyspnœa lasting a few or many hours, which, not infrequently, first appears during the night. Bronchitis usually accompanies the catarrhal attack, though this is not always described; at times the first dyspnœic paroxysm occurs after a prolonged attack of bronchitis, or it may show itself during a broncho-pneumonia. The dyspnœic attacks recur at intervals, though the catarrhal or bronchitic conditions may seem to subside, and the patient is now said to suffer from asthma. Sometimes asthma develops from chest troubles left by measles, whooping-cough, and other ailments, and very commonly it springs from the catarrhal condition connected with hay fever. More rarely it originates in quite a different manner; a patient is suddenly seized with an asthmatic paroxysm for which there is no certain cause to be found, though it may be attributed to emotional excitement, indigestible food and other antecedents. Asthma again has been known to arise suddenly during pregnancy.

The course of asthma is very variable; at times the attacks, when once established continue to occur at shorter or longer intervals throughout life, which nevertheless may be prolonged to the usual term, but if the attacks be frequent and severe, chest trouble often develops, which may shorten life. Sometimes after a few attacks the dyspnœa does not recur for long periods, or it may never recur. Sometimes the paroxysms lessen in intensity, alter in character, or quite disappear, even when they have been severe for years. Death during a paroxysm is extremely rare.

SYMPTOMS.—*The dyspnœic attacks* in asthma may develop without any premonitory symptoms, but patients are at times made aware of their approach in various ways, sometimes by a feeling of excitement or of depression, sometimes by undue drowsiness, more rarely by wakefulness.

Whether there are premonitory symptoms or not, the attacks most frequently develop in the night or early in the morning. A patient is often wakened by the advent of dyspnœa and finds himself "wheezing"; at times the dyspnœa only appears after waking. It may become pronounced within a quarter of an hour; more often it develops slowly. The patient may even have troubled sleep for an hour or two after the first symptom is felt, and many hours may elapse before the height of the paroxysm is reached. During the initial stage profuse diuresis sometimes occurs, and neuralgic pains may be felt in various parts; itching about the sternum, chin, and inter-scapular regions has been noticed. When the dyspnœa is fully developed, sleep is impossible; the patient either sits up in bed with head bent forward and the shoulders raised, or more often finds it necessary to rise and sit or stand in such a position that he may give the extraordinary muscles of respiration the best chance of acting. To fix his shoulders, neck, and back, he often plants his elbows or his hands on some piece of furniture and leans forward, raising up his shoulders with the head thrown back, mouth open, and nostrils dilated.

With anxious countenance and pale, sometimes slightly cyanosed face, he devotes himself entirely to his respiratory efforts, and often the face is bedewed with cold perspiration. The pulse is frequent and small, the temperature is usually normal. On examining the chest the first thing that strikes the observer is the vigorous action of all the extraordinary muscles of respiration, which are employed in raising the ribs and dilating the thorax, the fixed head, neck, and shoulders serving as points from which they act.

The chest is nearly always found in a condition of extreme inspiration, the diaphragm being depressed and the abdomen rendered full. A few observers have noticed the chest drawn in, wholly, or in part. The accessory muscles of respiration are seen in full action, yet they move the chest-walls very slightly. So little air enters the lung that it cannot follow this slight movement, hence the intercostal spaces are depressed as well as the suprasternal region and scrobiculus cordis.

The respirations are commonly slowed, but sometimes quickened, the expiration being almost always markedly prolonged, the inspiration following immediately on the cessation of the expiration. Rarely inspiratory dyspnœa with a raised diaphragm has been observed.

On percussion, the chest yields a hyper-resonant note, cardiac dulness is decreased, and the lung extends lower than in the normal state. Some observers, among them C. J. B. Williams and Walshe, have seen cases in which the resonance has been impaired.

On auscultation the respiratory murmur is lessened generally, and often annulled in some areas, rhonchi, chiefly sibilant, being heard all over or in many parts of the chest. The stethoscopic sounds not infrequently change much in the same areas during a paroxysm. The expired air contains much carbonic acid and no oxygen. After an hour or two, the severity of the paroxysm may pass off; rarely it ceases suddenly within that time; not infrequently the dyspnœa lingers on in a modified form until the middle of the day on which it has commenced, or it may last until the next day with exacerbations and remissions.

During the subsidence of the paroxysm, moist sounds may be heard over the lungs, and pellet-like opalescent masses of mucus are usually expectorated. But when the attacks are associated with catarrh, the expectoration raised may be in the form of large tenacious, sometimes frothy masses, or liquid and flocculent.

Blood is at times seen, and in long-standing cases the sputum may be muco-purulent. Plugs or casts, more or less twisted, may be seen with the naked eye in the sputum, and on microscopic examination, besides cylindrical epithelial cells, certain spiral bodies and crystals, to which v. Leyden first drew attention. The former, sometimes known as Curschmann's spirals, are most abundant in the pellet-like bodies. They vary in shape, but usually consist of a central thread round which is a meshwork of very delicate fibres, looped round in spirals, or occasionally retiform. They seem to be formed from a substance resembling mucin, and are believed to be due to twisting upon their axis of mucous threads. Berkart looks upon the outer spirals as developed from the central thread, but some observers think the latter is chemically distinct from the mucous spirals which surround it, and that it is of the nature of fibrin. The crystals which are said by Schreiner to be composed of the phosphate of a new organic base, the nature of which is not determined, are by some thought to be derived from the spirals. A. Fraenkel upholds the view that they are produced from the eosinophile cells. Asthmatic sputum contains a large number of leucocytes containing eosinophile granules, and it must be pointed out that none of these constituents are absolutely characteristic. The spirals were thought by Curschmann to indicate a special form of disease of the finer bronchial tubes (bronchiolitis), but they have been found in pneumonia and in pulmonary œdema, and similar bodies have been noticed in vascular keratitis. Crystals identical with those found in asthmatic expectoration are to be seen in post-mortem blood and some organic fluids, and eosinophile cells occur in the expectoration of acute and chronic bronchitis.

The dyspnœic paroxysms may last only half an hour to an hour, or more than twenty-four hours. After the urgency of the paroxysm has passed off there may be restoration to the normal condition, but occasionally a feeling of oppression remains for some time, a few rhonchi may be heard over the chest, and there is evidence of the imperfect entrance of air into some part of the lung. Attacks of asthma are at times slow in their advent, and slight both in duration and severity. They may be indicated merely by a short access of tightness of the chest and wheezing. Between these and such a typical paroxysm as has been described, there may be any degree of severity.

In the intervals between the attacks, asthmatic patients may be free from all discomforts or signs of physical disease, but this is only so when the attacks are of comparatively recent origin, or occur at long intervals. After the ailment has existed for some time, it commonly happens that shortness of breath and physical signs give evidence of chest troubles in the inter-paroxysmal periods; bronchitic attacks are liable to be induced by slight causes, or rhonchi may be constantly present. The symptoms and physical signs of emphysema are very commonly noted, and there may be dilatation of the right side of the heart. The confirmed sufferer from asthma often acquires, in consequence of his ailment, a characteristic aspect and physique. Where there are oft-repeated attacks, the patient has commonly an anxious look, the eyes are watery, the superficial veins are often conspicuous, the hands are cold and often blue, and especially when there is much dyspnœa between the attacks the face is slightly dusky. The confirmed asthmatic patient usually loses flesh. He has high shoulders and often stoops. If asthma occurs in early life, the lower part of the chest may be drawn in, a form of pigeon-chest being produced.

CAUSE AND FREQUENCY OF ATTACK.—Asthma is a periodic disease, the paroxysms recurring at intervals without cause. Nevertheless, certain conditions influence the advent of the attacks, and the character and time of food is perhaps the most notable of these. Indigestible articles of diet or a hearty meal taken late in the day will often produce in asthmatics paroxysmal attacks during the night, and not infrequently patients have to debar themselves of solid food after mid-day. Accumulations in the rectum may likewise cause paroxysms to occur. The inhalation of irritating substances has, too, a potent influence. Hay asthma is, of course, a well-known example of this, but in a few instances ipecacuanha has been found as powerful in its effect as the pollen of the *Anthoxanthum odoratum*. Two or three grains of ipecacuanha blown into a large room have been known to produce an attack in its occupant. The smell of cats, dogs, and other animals, produces asthmatic attacks in some. Atmospheric and climatic conditions are well known to influence sufferers from asthma. As a rule, the presence of fog, smoke, dust, and irritating gases, tends to induce asthmatic attacks; nevertheless this is not always the case, and some live more comfortably in a moderately impure than a pure air. It is well known that every asthmatic is affected in his own special way, by climatic conditions. A place which invariably brings on attacks in one sufferer from asthma may be found to suit another admirably. Exposure to cold winds is a common cause of attacks. Laughing, sneezing, or coughing may induce a paroxysm. Emotion has sometimes a marked effect on the attacks; a fright or a sudden pain may bring on a paroxysm, or more rarely cause one to cease. I have seen a typical asthmatic attack brought on by a subcutaneous injection of ergotine. Certain mental states seem to have some effect on the occurrence of paroxysm. Some patients going to a place where they have habitually had attacks of asthma are almost sure to have one immediately, but should they escape the first night they may remain free for a long period. Habit seems to influence both the occurrence and the form of asthmatic attacks.

It may be noted that the attacks seem to a slight extent protective; where asthma comes on at almost regular intervals an asthmatic is sometimes able to do those things immediately after an attack which will infallibly bring on another attack when the time for their recurrence is nearly due.

The advent of asthmatic attacks may be influenced by changes more or less evident in the conditions of various tissues and organs. A catarrhal condition, acquired or increased, tends to induce them. The nasal mucous membrane may become so altered as to be the main cause of asthmatic paroxysms. Disease of the uterus may render this organ the special centre whence arises the reflex-excitation leading to asthmatic attacks, and at times certain external parts of the body are so sensitive that even the application of cold may induce an asthmatic attack; on the other hand, the outbreak of a rash has been followed by the subsidence of asthma.

The paroxysms of asthma may, when uninfluenced by special causes, come on at regular or irregular intervals. They may occur, for example, every day, every week, every month, or at any other period of time; sometimes, though rarely, with absolute regularity, much more commonly the intervals are somewhat irregular, and not infrequently there is no definite time for recurrence.

Dr. Salter has pointed out that asthma paroxysms rarely occur daily unless associated with organic chest diseases, or unless the exciting cause recurs daily. A late dinner-hour may supply this exciting cause. Attacks not uncommonly come on once a week or once a fortnight. When attacks occur monthly in women they seem associated with menstruation. Asthma sometimes occurs only at certain times of the year, owing to special causes; in the winter only, for example, when complicated with bronchitis; when connected with hay fever, in the summer only; but sometimes it is limited to certain times of the year without known cause.

Morbid Anatomy.—Little is known of the nature of the morbid changes in asthma. Watson states that the bodies of asthmatic patients have often, on being examined, presented no vestige of disease in the lungs or heart, and Leyden says he has found the lungs of those who in early years suffered from asthma perfectly normal. Lung changes have indeed been recorded in post-mortems made on sufferers from asthma. Théry, in 1859, described the results of sixty-six autopsies on asthmatic people, but the changes noted in the chest were such as are commonly found in ailments with which asthma is usually associated, and throw no light on the condition present during the paroxysms. In recent years, however, the conditions observed on the post-mortem examinations of sufferers from asthma dying in a dyspnœic attack, have been carefully recorded in four cases.

Von Leyden, in a case complicated by emphysema, dropsy, and albuminuria, and dying in a prolonged paroxysm of dyspnœa with cyanosis and stertor, found at the autopsy no dilatation of the bronchi, but the bronchioles contained a little mucus, in which neither spirals nor crystals could be found, though both had been noted in attacks during life. Some of the alveoli were dilated, some normal; they contained granular masses, with a moderate amount of large cells; the walls of the bronchioles were not much altered, but layers of a coarsely granular, apparently amorphous, substance adhering to the surface of the mucous membrane reduced their calibre.

Berkart, in an old-standing case of bronchitis and asthma with anasarca and dilated heart, and dying after prolonged dyspnœa and cyanosis, found in the right main bronchus a loose cylinder, one branch of which extended into a bronchiole, and in the left main bronchus, in addition to a similar formation, a little greenish mucus.

Microscopically the cylindrical mass contained cylindrical epithelium and spirals, but no crystals. There were polypoid excrescences on the right bronchus, fibroid in character with some ulceration. The smallest bronchi were dilated and denuded of epithelium; they contained masses of exudation almost made up of colonies of streptococci. In the bronchi of medium size there was hyperplasia of the mucous membrane, and they contained masses of detritus, with a few fragments of crystals. The alveoli were dilated, and some of them were nearly filled with fibrinous exudation.

Schmidt describes the condition of the bronchi in the case of a patient who suffered from asthma as well as cancer of the lung, and who died in a paroxysm. The bronchi of the upper lobes of both lungs were filled out with gluey tenacious mucus which could be drawn out in long spiral threads. In the lower lobes of both lungs the bronchi near the hilus contained some plugs of moderate length and slight consistency. The microscopic examination showed that in the majority of the small widened bronchi the epithelium was in good condition, but the connective tissue beneath was infiltrated with round cells. The contents of the bronchi consisted chiefly of mucus in which various cell elements, alveolar epithelium, leucocytes, and degenerated cells, round, oval, and spindle-shaped, were embedded. Spirals could be traced into the smaller bronchi, not into the alveoli.

A still more important case is recorded by A. **Fraenkel**. A gouty, bronchitic, and emphysematous man, who had suffered from asthma for two or three years, died during a paroxysm. The bronchi were dilated, and their mucous membrane markedly reddened and filled with tenacious material. No typical spirals could be detected in it; but it contained some threads, apparently fibrinous, and abundant ciliated epithelium. The middle and small bronchi showed marked desquamative catarrh, the cylindrical epithelium was raised over large areas from the underlying tissue and completely blocked the widened lumen of the tube. Elongated epithelial cells abounded on the surface from which the epithelium had been cast. There was considerable increase of interstitial and inter-alveolar tissue. Infiltration of lymph cells into the walls and lumen of the bronchi was noticed, and here and there it appeared as if pus were forming.

Nature and Etiology.—The post-mortem appearances described do not disclose either the exact condition of the lung tissues which actually causes the attack, or the real nature of the ailment. On the former point many opinions have been entertained, which may be divided into three classes.

(1) The view that spasm of the bronchial muscles is the immediate cause of the asthmatic paroxysm has longest held the field. Willis, as early as 1670, promulgated the opinion that what we now know as asthma is a spasmodic or convulsive ailment due to spasm of the air-tubes; and Floyer, in 1698, regarded contraction of the bronchial tubes as the cause of asthma. The discovery of Reisseissen of the bronchial muscles seemed to explain the asthmatic paroxysm, but for a long time there was great doubt concerning the action of these muscles. In 1840, C. J. B. Williams showed that the bronchi contract on stimulation, but he did not discover the action of the

vagus on them. This, however, was shown by Longet and Volkmann, and Roy and Graham Brown have definitely shown that stimulation of the vagus is capable either of dilating or contracting the bronchi.

Williams, Hyde Salter, Wilson Fox, and Thorowgood, in this country, and Laënnee, Trousseau, Riegel, and Biermer, on the Continent, may be mentioned among those who have attributed asthmatic attacks to bronchial spasm. Williams and Walshe held the same view, but also thought that paralysis of the bronchial muscles might, under some conditions, give rise to asthmatic paroxysma.

With regard to the method by which contraction of the bronchi causes the symptoms and physical signs of asthma, opinions have also differed. Biermer has suggested that the contraction of the bronchi is more readily overcome by inspiration than expiration. Hence the lung becomes distended, for the bronchi are sufficiently dilated during inspiration for air to enter through them, which the expiratory power is insufficient to expel. It can be easily understood how, under these conditions, dilatation of the air-cells can take place, and how, too, occasionally, lung collapse may occur, when bronchi are so contracted that they do not become sufficiently expanded during inspiration to admit a due supply of air. Biermer has suggested, too, that after long contraction the muscles may lose their power, and that this may lead to emphysema.

(2) Wintrich and Bamberger have expressed the opinion that spasm of the diaphragm plays the chief part in the production of paroxysmal attacks, whilst Budd, Jaccoud, and G. See have attached more importance to spasm of the respiratory muscles. It has been held by Lebert that diaphragmatic spasm follows and is to some extent induced by spasm of the bronchi.

(3) Asthmatic attacks are commonly, possibly always, accompanied by changes in the bronchial mucous membrane, but very divergent views have been expressed with regard to the relation of these changes to the attacks. Biermer has suggested that the bronchial spasm may cause alterations in the mucous membrane, though he also holds that fluctionary hyperæmia may be primary. A. Fraenkel inclines to the view that asthmatic attacks are due to bronchial spasm, but that concomitant changes of various kinds take place in the mucous membrane. The epithelium exfoliates, the spasm aids the process, and the cast-off epithelium blocks up the air-passages.

An English physician named Bree, writing in 1807, attributed asthma to the irritating nature of the contents of the bronchi, and held that the spasmodic attacks were due to the efforts of the lung to remove the cause of irritation. Views somewhat allied to this have been promulgated by Leyden and Curschmann. The former thinks that asthmatic attacks are due to material transuded into the cells and bronchi, and that the crystals developing in it play some part in causing bronchial spasm. The latter assumes the existence of a special affection of the bronchial mucous membrane, bronchiolitis exudativa, leading to the same result.

Several French writers, Rostan, Beau, and Louis in the early part of the century, also looked upon asthma as due to catarrhal affection of the bronchi. Very recently, in his Lumleian lectures, Gee has expressed a similar belief. He holds that asthma is merely a peculiar form of bronchitis, and that there is no need for any hypothesis of spasm in order to explain the phenomena of the disease. Berkart, too, discards all idea of bronchial spasm, and regards asthma "as an acute paroxysmal, nay, almost erysipelatous form of inflammation, which extends from the pharynx upwards and downwards, and is accompanied by a croupous exudation."

Weber attributed the symptoms of asthma to tumefaction of the bronchial mucous membrane from dilatation of the blood-vessels through vasa-motor nervous influence, and Sir Andrew Clark suggested that they are due to congestive swellings of the mucous membrane coming and going with greater or less rapidity, and analogous to the condition of the skin in nettle-rash, the swelling subsiding after free secretion. He recognised, however, a nerve discharge as the cause of these congestive swellings, and he thought the contents of the bronchi might excite some contraction of the bronchial muscles, but not sufficient to account for an asthmatic paroxysm.

It is not possible to give the arguments which have been adduced for and against the views thus set forth. The theory that bronchial spasm is the chief cause of the phenomena of the asthmatic paroxysm has most evidence in its favour, and certainly has not been rendered untenable by the arguments used against it. It receives support from the nature of the remedies found to relieve asthma. Chloral-hydrate and morphia, which remove spasm, are markedly beneficial, and the nitrites and atropine are largely used to allay the paroxysms. Now the nitrites relax the contraction of involuntary muscular fibre, and Roy, in describing the influence of stimulation of the vagus on the bronchial muscles, points out that atropine causes a complete paralysis of the constricting fibres or of their terminal apparatus in the bronchi, so that after a very small dose, stimulation of the peripheral end of the cut vagus, which has previously caused strong contraction, no longer has any effect. Whatever may be the seat of the changes which immediately give rise to an asthmatic attack, there can be little doubt that disturbed innervation is the chief factor in causing asthma. The occasional alternation of asthmatic attacks with epilepsy, some forms of insanity, and various neuroses, the induction of paroxysms by emotion, and the influence which habit and expectation have at times on their occurrence, seems to show that the disturbance may have a centric cause. It seems clear, too, that in a very large number of cases the peripheral nerves participate in or are the main seat of the disturbance. This is shown by the curative effect which has been known to follow the removal of a polypus from the nose, and by the cessation of attacks which has been recorded on relieving pressure upon the vagus. It seems very probable that the periphery of the vagus in the bronchi is not infrequently the special seat of irritation. But the conditions found after death in a paroxysm seem to indicate that desquamation and other changes in the bronchi play a part, in some cases at least, in the production of dyspnœa.

Asthma is commonly made worse by catarrh. At times the disturbed innervation renders areas susceptible to certain irritants only. Thus, asthma may be caused in one by the pollen of grass and not by smoke, whilst in another exactly the reverse may be the case. Probably in many cases there is disturbed innervation both general and local. It may be that the muscles of the bronchi themselves are at times unduly susceptible to reflex irritation, but of this there is no absolute proof. There can be little doubt that the altered condition of the bronchi has in many cases some share in causing the dyspnœa.

Asthma seems in some cases hereditary, and neurotic inheritance doubtless plays a part in predisposing to it; so does sex. Males are more liable to asthma than females. Childhood is the period at which it most frequently appears, but it may come on at any age. Gout and syphilis and other disorders have no doubt a predisposing influence, though the method in which they act is obscure. It would seem as if some morbid nutritional influences may expend their energies in producing at one time the peripheral disturbances essential for asthma, at another time in causing eczema or some other cutaneous affection, for cases are recorded in which asthma and such affections have appeared alternately.

Diagnosis.—It sometimes happens that paroxysmal attacks due to laryngeal changes are mistaken for asthmatic attacks, but the stridor and the inspiratory character of the dyspnœa, usually present in laryngeal affections, will, as a rule, suffice to prevent a mistake of this kind. Physical examination will distinguish between hysterical attacks simulating asthma and the real ailment. Fibrinous bronchitis will not present much difficulty, but the dyspnœic attack which accompanies the casting off of the bronchial casts in this ailment might lead to errors unless the sputum be examined.

The diagnosis of asthma from paroxysmal uræmic and cardiac dyspnœa is usually easily made.

The gasping breathing, more or less periodic, occasionally seen in uræmia, can hardly be mistaken for asthma. Very rarely, however, paroxysms of dyspnœa occur in chronic Bright's disease, which cannot be distinguished from genuine asthma and probably own the same cause. In cardiac dyspnœa, which is due to the influence of a disordered circulation on the lungs and nervous system, the breathing is more panting, and generally increased in frequency; the expiration is not prolonged, and there is an absence of that want of correspondence between the efforts made to dilate the chest and the entrance of air which is characteristic of asthma. But in cardiac diseases doubtless true asthma is at times present in connection with the condition of the heart. It may be that, as Kingscote suggests, an enlarged heart may, by pressure, cause vagus irritation and true asthma. Mediastinal tumours and aneurysm of the aorta cause at times paroxysmal dyspnœa, but the concomitant symptoms will usually prevent this being mistaken for asthma.

The diagnosis of bronchitis with dyspnœic attacks from asthma complicated with bronchitis and emphysema is more difficult. Nocturnal exacerbations of bronchitic dyspnœa are not uncommon, and at a late period of asthma the symptoms of bronchitis and emphysema may be very pronounced in the inter-paroxysmal periods. Moreover, it is probable that such disturbances in the peripheral nerves of the bronchial mucous membrane may be induced by the changes consequent on chronic bronchitis, that a condition leading to genuine asthmatic attacks may be induced. The history of the ailment is to some extent a guide to the diagnosis. If in the early stage there has been paroxysmal dyspnœa without indications of lung or heart disease, the nature of the case cannot be doubted, even though later on bronchitic symptoms are prominent. When such history is wanting, and the attacks have supervened on prolonged bronchitis, the diagnosis may be doubtful in some cases where the dyspnœic attacks are slight and bronchitis much in evidence. The nature of the expectoration may aid in the discrimination between asthma and ailments simulating it. If the sputum contains spirals with a central band or Leyden's crystals, with cylindrical epithelium and a considerable quantity of eosinophile cells, the presence of true asthma is probable, even though, as before stated, all the various substances have been found present in the sputum in other ailments.

Treatment: (*a*) *Preventive.*—For the cure of asthma the removal of certain conditions on which it depends is the first point to be aimed at. It is at times possible to annul the peripheral irritation which causes the ailment, either by acting on the area where the peripheral nerves are unduly sensitive, or by abolishing or lessening the strength of the irritant which acts on the area. It is perhaps possible, too, to cure asthma by decreasing that form of general excitability of the nervous system on which it seems that asthma at times depends.

The mucous membrane of the nose and bronchi require careful attention. Voltolini recorded a case in which the removal of a nasal polypus was followed by a cessation of asthma, which recurred when the polypus again appeared, and cases have been published since then in which the cure of an irritable condition of the mucous membrane over the middle turbinated bone, or removal of the bone itself, has led to the cessation of asthmatic attacks. A careful examination of the nasal mucous membrane and the septum should be made in cases of asthma, with the view to the removal of any abnormal condition. Even if any condition of importance be present, it is impossible to predict that any beneficial result will follow its appropriate treatment. M'Bride has pointed out that those cases where the application of a probe to a localised sensitive area in the nasal mucous membrane induces cough as a reflex, offer a more hopeful prognosis as to the result of local treatment. This subject will be further considered under "Hay Fever."

The state of the bronchi, too, often requires attention. Though asthma may exist apart from manifest changes in the bronchial mucous membrane, yet whatsoever evilly influences the condition of this membrane, tends not only to the increase of the violence of the paroxysms, but also to the continuance of the ailment, and everything which improves its condition tends to cure asthma. Hence catarrhs must be avoided by due care with regard to exposure to cold by warm clothing, etc., and bronchitis must be lessened where possible, by expectorants and other forms of medicinal treatment into which I need not here enter.

The inhalation of specially irritating substances, whether it be the pollen of grass, dust, or smoke, must be avoided as far as possible, and careful enquiries should be made in every case for undiscovered idiosyncrasies.

Many years of suffering have been caused owing to the fact that the influence of an emanation from an animal or from a plant has in some the power of causing asthma, and the possibility of an idiosyncrasy of this kind should always be remembered.

A suitable climate must be sought for the sufferer from asthma. As is well known, there is no one place which is specially suitable for asthmatics. Some find the sea air suits them best, some can only live comfortably in the country, some like a dry air, others are only well when it is moist. The suitable place for each asthmatic patient can only be selected by trial. It is somewhat remarkable that the air of London is very commonly found advantageous. Almost every place on the coast of England has been found at one time or other beneficial to asthmatics, and almost every place has been found detrimental to individual cases. The same thing is true of continental places. I have known patients who have found their asthma absolutely cured by a residence at Les Avants, and others who were scarcely able to breathe there.

Irritation of the gastro-intestinal tract must be dealt with, and diet should be carefully attended to. The presence of indigestible substances in the stomach will in many ensure a severe asthmatic paroxysm, and a loaded rectum may have the same effect. A useful rule is to take small meals of one course only, the mixture of meat food with farinaceous food or articles of diet containing much sugar is often injurious. The time at which food is given requires special notice. A heavy meal after mid-day will in many asthmatics ensure a paroxysm in the night.

Attention to the condition of the heart will sometimes lead to the cure of asthma. It has been suggested that by decreasing the size by treatment, less pressure is exerted on the vagus behind the heart, and therefore that paroxysmal attacks dependent on the irritation of the vagus may thus be cured. Caffeine is sometimes found to have a considerable influence in the cure of asthma; it is probable it acts by its favourable effect on the heart. It is used sometimes for the relief of asthmatic paroxysms.

Although it is not proved that asthma is actually caused by a general disturbance of the nervous system, yet the influence which some alteratives and tonics have in the cure of asthma renders this probable. Iodide of potassium has in many cases a markedly beneficial influence, which is specially noted in those cases in which a certain amount of continuous bronchitis is accompanied by nocturnal paroxysmal attacks of dyspnœa. Five to ten grains of the iodide given three times a day, will very commonly give great relief, and at times, under its influence the paroxysmal attacks completely disappear. Arsenious acid in the form of five drops of Fowler's solution three times daily is at times beneficial, and in a few cases zinc salts are reported to have done good. The hypophosphites of soda and lime, too, in doses of four grains, three times a day, have proved of advantage in many cases. The adoption of these means, medicinal or otherwise, which are calculated to improve the general health, are likely to be of service, and ailments such as gout and syphilis must be met by appropriate treatment. It is worthy of note that occasionally members of gouty families are attacked by asthma in adolescence, and that there is probably a connection between asthma thus occurring and the hereditary gouty influence.

(*b*) *Treatment of Paroxysm.*—Whilst adopting means for the cure of asthma, *i.e.* for the prevention of the attacks, the alleviation of the paroxysms also requires attention. It has been already pointed out that the substances that are commonly used for the relief of asthmatic paroxysms contain either a nitrite, or atropine, or one of the allied alkaloids. Nitrites are used both internally and by inhalation. The action of nitrite of amyl when inhaled, is of such short duration that it is only occasionally that it suffices to check a paroxysm. Nitrite of sodium and nitroglycerine (which acts like a nitrite) are sometimes advantageously employed, but the solution of nitrite of ethyl of the pharmacopœia is the most convenient, and at the same time the most effective, nitrite for administration. Two drachms of the solution should be given in two tablespoonfuls of water, and afterwards a drachm every ten minutes for three doses. Belladonna and stramonium are sometimes of great value when given internally in bronchitis with paroxysmal dyspnœa, they should be given in full doses, ten to fifteen minims of the tinctures. Where the paroxysms come on suddenly and at long intervals they will not be found of value.

For the immediate relief of asthmatic attack the smoke derived from the burning of a large number of substances in the form of powders and paper, is much used, and many forms of cigarettes are likewise sold. Out of eighteen such substances which were tested, the vapour of eight was found to contain a nitrite, and in that of thirteen there was distinct evidence of a pupil dilator. There can be no doubt that the inhalation of the vapours from the substances above mentioned are at times of temporary service, but no one can say which will act the best in any individual case. They all may be tried in turn, but it must be remembered that excessive use of some may lead to the production of the physiological effects of the mydriatic substances contained in them. Injections of tartrate of morphia, gr. $\frac{1}{6}$-$\frac{1}{4}$, are of great value in relieving paroxysms of asthma, but when the asthma is complicated with much bronchitis it becomes less suitable, and must be used with great care. Chloral-hydrate in doses of twenty grains will also cut short spasmodic attacks, and, provided there is no evidence of cardiac failure, may be given in cases where there is much bronchitis present. The compressed-air bath when available gives relief, and its continued use seems in some cases to render the asthmatic attacks milder.

Remedies which induce nausea and depression are undoubtedly sometimes of service in cutting short a paroxysm of asthma. Tobacco smoke to excess so as to cause nausea is sometimes effective in shortening an attack. Lobelia probably acts by the nausea and depression it produces. In ordinary asthma it has not proved in my hands of much service. The inhalation of the spray of ipecacuanha wine is a remedy more adapted for the paroxysms of dyspnœa which sometimes accompany bronchitis than for genuine asthma, and the same may be said of ammonia, the vapour of which, carefully administered, gives great relief in some forms of bronchitic dyspnœa. It would probably fail entirely, however, in cutting short a paroxysm of asthma. Pyridine has been used with success, but its odour is very objectionable. The inhalation of chloroform has been found of some service in relieving dyspnœa during asthmatic attacks, and iodide of ethyl is also beneficial. Ether is not as a rule found beneficial, though the administration of ether in the form of perles or capsules is at times advantageous in the dyspnœa of chronic bronchitis. Acetic ether, too, is at times of value, both for internal administrations and inhalations. Hot coffee seems to do good, so, too, does alcohol, but it is not advisable to recommend patients to resort to alcohol for the relief of dyspnœic paroxysms.

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Astigmatism. *See* REFRACTION.

Astragalus. *See* ANKLE-JOINT.

Astringents.—*Definition.*—An astringent substance is one which can coagulate or contract the protoplasm, and consequently lessen the activity, of living cells and tissues, diminish the calibre of the arterial vessels, or cause the coagulation or precipitation of albuminous bodies in fluids. Astringents decrease cellular, secretory, and excretory activity, and diminish exudation.

Hæmostatics and styptics are terms applied to astringents which possess marked powers of checking loss of blood. Various drugs act indirectly after the manner of astringents in lessening secretion or excretion, as, for instance, those which diminish the peristaltic movements of the intestinal walls.

Modes of Action.—(*a*) Drugs which constrict the blood-vessels either generally or locally. Ergot, digitalis, and hamamelis are examples of drugs which cause vascular contraction by acting upon the vaso-constrictor centre; dilute sulphuric acid, alum, and the application of cold may be taken as types of those acting locally.

(*b*) Drugs which coagulate proteids in the blood or the cells. Lead, mercuric, copper, silver, and ferric salts; tannic acid, and drugs containing it; strong alcohol, hot water; carbolic and mineral acids in strong solutions, or pure. The areas of action influenced by the local application of these remedies vary in direct relationship with the nature of the coagulum produced. Thus the action of nitrate of silver extends over only a very limited range, owing to the formation of an inert silver albuminate; a property which is made use of in treating corneal affections, the thin skin of silver albuminate following from instillation of its solution protecting the cornea from chance of injury by further action. The iron per-salts act more widely; while hydrochloric and sulphuric acids in strong solution are able to redissolve the coagula formed by their action, and therefore may influence the deeper parts, nitric acid and glacial acetic acids are not able to do so, and exert a more circumscribed effect.

The new synthetic compounds of tannic acid with various organic substances, such as tannoform, tannopin, tannalbin, tannigen, and others, are credited with very favourable properties as intestinal astringents. Compounds of proteid and silver salts such as protargol are likewise of service in many conditions.

As alcohol only precipitates and does not coagulate the proteid constituents of the tissues and blood, the precipitate caused by its application may shortly be redissolved; its local action, therefore, is but temporary.

(*c*) Remedies acting in a physical manner.

Matico leaves, in virtue of their hairy surface, lycopodium giganteum, the puff-ball, cobwebs, cotton-wool, and other substances presenting a finely divided texture, act as astringents (styptics or hæmostatics) by facilitating coagulation of the blood; the contraction in bulk following on this constricts the blood-vessels.

(*d*) Drugs exerting a local action on the tissues when given internally, but which do not cause contraction of the vessels.

Tannic acid and alum when given by the mouth do not contract the blood-vessels, but when absorbed into the blood act as astringents, and must therefore affect the tissues themselves.

(*e*) Drugs having an astringent action upon the alimentary canal.

The foregoing groups of astringent drugs act in the same manner upon the stomach and bowel; by contracting the vessels and by coagulating or precipitating the cell-proteids. Other drugs diminish peristalsis—opium, chalk, and lead acetate—or decrease glandular secretion, and thus act in a manner which brings them within the group.

Among the astringents which are of especial value in relation to the alimentary canal, but which seem to be inert, or at least act as inert bodies when applied externally, the salts of bismuth and oxalate of cerium may be cited. Their mode of action is obscure; the results of their action are very variable, but sufficiently often so beneficial that they must be accorded some astringent properties.

Uses.—The most immediate uses to which astringents, including under the term styptics and hæmostatics, are put, undoubtedly consist in the arrest of hæmorrhage and of diarrhœa. Hot water and pressure may suffice to check hæmorrhage from accessible parts, if ineffectual, perchloride of iron in solution, nitrate of silver, the application of lycopodium, etc., can be tried. If the hæmorrhage is internal, ergot, morphina, iron per-salts, the application of cold externally, and the adoption of absolute rest, may be successful. In hæmorrhage from the stomach or bowels, local astringent action may be obtained from tannic and sulphuric acids, the oxides of silver and zinc, acetate of lead, alum, ferric salts, or sulphate of copper.

Ergot, morphina, hyoscyamus, and lead salts may also check the bleeding by their more general actions.

In gastric ulcer a dilute solution of nitrate of silver (1 gr. to 1 oz.) often appears to act beneficially; and in dysentery, massive rectal injections of the same solution are often successful. For the arrest of diarrhœa little need be added to what has already been noted. The probable origin of the diarrhœa should be borne in mind when making choice of the astringent remedy, *i.e.* whether it be vascular, glandular, peristaltic, or inflammatory.

In many instances dilute solutions of substances, which, when in more concentrated form, act as corrosives and escharotics, prove to be most valuable astringents. Care must be taken, however, in making use of such solutions, to avoid any untoward results which may follow the application to one individual of a lotion innocuous to many others, but, owing to personal idiosyncrasy, injurious to him. This occurs most frequently when solutions of mercuric or lead salts are employed; we have met with a case in which one painting of the pharynx with 1-480 perchloride of mercury solution with a by no means full brush, was followed by poisonous sequelæ.

Atavism. *See* HEREDITY.

Ataxia. *See* EQUILIBRIUM.

Atelectasis. *See* LUNGS.

Atheroma. *See* VESSELS, DISEASES OF.

Athetosis.—DEFINITION.—A term introduced by Hammond in 1871, and applied to certain cases "mainly characterised by an inability to retain the fingers and toes in any position in which they may be placed, and by their continual motion." The movements are spontaneous, deliberate, and monotonous in character, and are most marked in the fingers, and it may be the toes, of one side or of both.

ÆTIOLOGY.—Athetosis is simply a symptom, associated with divers lesions of the brain. The ætiology, accordingly, is somewhat varied. One group of cases is congenital, athetosis being a common symptom in the cerebral birth palsies which result from injury to the brain during labour. These cases are usually bilateral, and the paralysis is associated with spasm. The children are usually idiots.

In early childhood, athetosis may occur in connection with hemiplegia. Infectious diseases, and especially whooping-cough, play an important part in the etiology. The condition commonly follows an attack, or a series of attacks, of convulsions. Mental defects are common.

In adults the condition is commonly associated with hemiplegia, the athetoid movements appearing in the affected limbs during recovery from the paralysis. In other cases there is an alcoholic history, and the movements appear after a series of epileptic fits.

In very rare cases the condition appears to be directly due to injury.

SYMPTOMS.—The mode of onset varies according to the etiology of the case. The condition may set in suddenly without preceding symptoms, or after symptoms of so slight a nature as to have been overlooked.

The movements usually occur on one side only, but in children most of the cases are bilateral. The fingers are seen to be in continual movement of a slow, monotonous, rhythmical character, now spread wide apart, now brought together, flexed, extended, hyper-extended. The wrist also may be flexed and extended, and the forearm pronated and supinated. Usually the movements are confined to the lumbricales and interossei, and it may be to the long extensor of the fingers. The flexion of the fingers is usually at the metacarpo-phalangeal joints, the second and third phalanges remaining extended. In children hyper-extension may be so marked that subluxation of the second and third phalanges may occur.

The toes are usually affected to a less degree than the fingers.

In rare cases the face is also affected. The voluntary movements of the face may be exaggerated on the affected side. Tremor of the face and tongue have been noted, probably in some instances alcoholic in origin.

In some cases the movements continue during sleep, in others they do not.

ASSOCIATED PHENOMENA.—As a rule the patients complain of no pain or fatigue. There may be associated rigidity (*e.g.* of the arm or of the calf) of the affected side.

The affected muscles may be somewhat wasted; or they may be of normal size; or they may be hypertrophied, even greatly hypertrophied, from the constant movement. The movements never occur in limbs completely paralysed. A degree of voluntary power is always present. Voluntary efforts, as a rule, increase the spasm. Thus Hammond narrates of his first case, "When told to close his hand, he held it out at arm's length, clasped the wrist with the other hand, and then exerting all his power, succeeded, after at least half a minute, in flexing the fingers, but instantaneously they opened again and resumed their movements." Sensation is often impaired. In cases following hemiplegia there usually is, or has been, associated hemianæsthesia. In children the sensory functions are usually normal.

The mental condition varies greatly. Some degree of mental impairment is very common in young children, amounting even to idiocy.

PROGNOSIS.—The prognosis is unfavourable. In many cases the movements continue without alteration for years.

DIAGNOSIS.—The diagnosis depends on the character of the movements, and can seldom present any difficulty. Athetosis has to be distinguished from other forms of post-hemiplegic movement, and from the movements of chorea, disseminated sclerosis, and paralysis agitans. A hysterical athetosis has been described.

The various post-hemiplegic movements are evidently closely related, and the different groups usually described—simple tremor of the affected muscles, quick incoördinate chorea-like movement, and athetosis with and without associated rigidity—cannot be sharply defined.

Morbid Anatomy and Pathology.—Athetosis is a symptom of disturbance of function, rather than of destruction, of part of the motor tract, and the result of post-mortem examinations, of which a considerable number have now been recorded, has been to show that in the majority of cases the lesion is situated in the neighbourhood of the basal ganglia, and especially in the optic thalamus and lenticular nucleus,—a result quite in accordance with what had been considered probable from clinical and experimental evidence.

As regards the nature of the lesion, cases occurring in adults are more often the result of softening (*e.g.* embolic) than of hæmorrhage, and in such cases slight damage to the cerebral structure is probably more extensive than the actual lesion. Thus a lesion apparently confined to the optic thalamus might interfere with the function of the adjacent internal capsule.

Another explanation of cases where the lesion is outside the actual motor tract is that the cerebral cortex may be interfered with indirectly by disturbance of function.

In a certain number of cases the lesion may actually be in the cortex, as in an instance given by Demange where hemiplegia had existed for thirty-three years. An extensive cortical lesion was found involving all the motor area behind the fissure of Rolando. The basal ganglia and internal capsule were healthy.

Clinical evidence to the same effect is supplied by a case of Beevor's, in which, immediately after an injury to the head, athetotic movements appeared in the right side of the face and right thumb, later in the fingers, and later still in the lower limb. In this case the nature of the injury and the march of the symptoms seem to indicate a cortical lesion.

In children the lesions are those of infantile cerebral paralysis. Cortical sclerosis and porencephalus are the most important conditions found post-mortem. In many cases the damage is very extensive, and the basal ganglia are involved. These conditions may be the result of a primary vascular lesion. Strümpell's theory of a primary polio-encephalitis has not been very generally accepted.

TREATMENT.—Treatment is very unsatisfactory. Gowers has seen improvement result from galvanism, but in other cases galvanism has been used for months without effect. Nerve sedatives, such as the bromides, and nerve tonics, such as arsenic, phosphorus, cannabis indica, may be tried. Nerve-stretching has been reported as giving a favourable result.

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Atlanto-axial Disease. *See* SPINE.

Atrophy.

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ATROPHY is defined as a diminution in the size of the structural elements of an organ or tissue. In accepting this definition it should be noted that we may find atrophy of the elements associated with enlargement of an organ, as in emphysema.

PHYSIOLOGY OF TROPHIC INFLUENCE.—If the function of a mixed nerve-trunk be seriously impaired by artificial section, injury, inflammation, or other cause, in addition to the disturbances of motor power and sensibility induced, definite changes ensue, due to loss of trophic influence. The nerve-fibres lose their excitability and degenerate; the muscles become soft and atrophy; the skin loses its elasticity, becomes thin and dry, due to impaired functional activity of its glands, and changes due to disturbances in the blood and lymph circulation—coldness, œdema, congestion—become apparent. The explanation of these phenomena was first afforded by the following experiments (Waller):—

1. On section of the anterior (motor) root of a spinal nerve, the peripheral end was found to undergo a simultaneous degeneration throughout its whole course, degenerative changes being visible in the axis-cylinder, medullary sheath, and neurilemma of the nerve; the central end of the nerve did not degenerate. The explanation is that the cells in the anterior cornua of gray matter exercise a trophic influence on the outgoing nerve-fibres, as well as on the tissues supplied by them.

2. On section of the posterior (sensory) nerve-root, different results ensued, according as the nerve was divided in the central or peripheral end of the intervertebral ganglion. (*a*) When the nerve-root was divided between the cord and the ganglion, the central end degenerated, the degeneration extending along the cord as far as the medulla; the peripheral end remained unaffected. (*b*) When the nerve-root was divided on the distal side of the ganglion, the peripheral end underwent simultaneous degeneration out to the periphery; the central end remained unaltered.

The explanation is that the cells in the posterior root ganglion exercise a trophic influence (1) on the sensory fibres coming from the periphery, and (2) on the ascending sensory fibres in the cord as far up as the medulla. The exact nature of this "trophic influence" is still unknown.

VARIETIES.—Atrophies are usually classified as congenital or acquired, physiological or pathological, general or local. The term "congenital atrophy" is an inaccurate and misleading one, as the conditions described are really examples of arrested growth, and are dependent on some perversion of innervation or obstruction to the blood-supply during development.

A. PHYSIOLOGICAL ATROPHY.—There is no more interesting study than the changes which the system undergoes in passing from the state of maturity to that of old age. These changes are regulated by a general law in physiology, which indicates that all organs attain their maximum of development at the period when their functions are most exercised, and subsequently decline as their functions cease to be required in the economy. The influence of diminished vital forces, as well as diminished functional activity, on the structures of the various organs and tissues, is an important and far-reaching one, and a study of the facies and bodily and mental movements of an old man form a striking object-lesson that "all cells are doomed to perish by the slow but unerring operation of natural causes inseparable from their nature and coeval with their birth." When the forces of disassimilation attain an increasing preponderance over those of assimilation, nutrition is diminished in proportion, and atrophy ensues. The resulting changes may be briefly summarised as follows:—

1. *The proportion of fluid* in each organ and tissue is diminished, the tissues tending to condensation. This is well seen in the muscles (*cf.* the flesh of a chicken with that of a "boiling fowl"), subcutaneous tissues, arterial system, brain, and other organs.

2. *Glandular activity* progressively diminishes, *e.g.* the skin, thyroid, and

various internal secretions. This accounts for the impaired digestive capacity of later life.

3. *The elasticity* of certain tissues disappears, notably the skin, muscular and arterial system. The flaccid and wrinkled skin of old age depends on its loss of power of accommodating itself to the size and figure of the parts it covers.

4. *The muscular system*, perhaps as a result of the condensation before referred to, loses tone, and as a result we find the various functional disorders of the heart, bladder, and other muscular tissues that are commonly met with as age advances.

5. Associated with the diminished secretory activity there is an increased functional *activity of the absorbent system*, evidenced by the hollow cheeks, sunken eyes, and the degree of general emaciation incident to that period of life.

6. *The nervous system* takes a prominent part in the retrogressive change. The garrulity of old persons, for example, may be regarded as an atrophy of memory. The general condensation of tissue previously referred to is well seen in the brain and cord, but it is impossible to say how far the neuro-muscular changes referred to are in any way causal of the other conditions mentioned.

Apart from the atrophy associated with old age, other examples of physiological atrophy occur. Of these the most pronounced are the atrophy of the thymus in early life, and, in adults, the involution of the uterus after parturition.

B. PATHOLOGICAL ATROPHY.—Whatever may be the real nature of the atrophic change which constitutes the natural process of decrepitude, decay, and death, it is a process only too seldom seen. The "age that melts in unperceived decay" is comparatively rare, owing to the ravages of the numerous diverse diseases to which man is liable, and which furnishes us with so many instances of premature decrepitude or pathological atrophy. This may be a general or a local condition, the nature being similar in either case.

Nature.—Atrophy consists in the diminution in size and number of the proper tissue elements, with increase of the interstitial connective tissue, a fibrous degeneration. A partial obliteration of blood-vessels frequently occurs in the process, and this further aggravates the atrophic change. Pigmentary changes are frequent, more especially in nerve and muscle cells. Fatty changes are also present in the liver, kidney, heart, or other muscle, and other alterations of the character of the protoplasm, evidenced by their altered staining reactions. These changes are usually most pronounced in the more highly specialised part of the cell, that is, the nucleus and its neighbourhood.

General atrophy or wasting is a familiar clinical picture arising from many diseased states, perhaps the most typical illustration being seen in cases of long-standing malignant disease in some part of the alimentary canal, or in cases of tuberculosis. From whatever cause arising, definite atrophic changes are seen in most of the organs and tissues of the body. The fatty and muscular tissues are first affected, then follow the blood and blood-forming organs, more especially the bone marrow, the nervous and bony systems being the last to show evidence of the atrophic change. It is important to note that in addition to the numerous cases of general atrophy resulting from *defective nutrition from without*, others occur when the defective nutrition is due to *faulty metabolism within*, one of the most striking examples of the latter being diabetes mellitus. The special form of general atrophy or marasmus seen in infants is described under "Atrophy, Infantile."

Local Atrophy.—The immediate nutrition of any tissue depends on the following four factors:—

(i.) The influence of the central nervous system (brain and cord).

(ii.) The state of the circulation, including the condition of vessel wall and of vaso-motor tone.

(iii.) The condition of the blood.

(iv.) "The trophic energy of the tissue."

The last factor, being dependent on hereditary qualities, is a varying one in different individuals, and is of considerable importance, and it, in turn, is largely influenced by the condition of the general trophic system.

Almost all cases of local atrophy are due to a disordered state of one or other or all of these factors, and in the investigation of every case the examination should be conducted along these lines. By reference to them a diagnosis of the cause can, as a rule, be readily made. Thus we find atrophy of groups of muscles in progressive muscular atrophy, poliomyelitis anterior acuta, transverse myelitis and other similar lesions, due to the loss of the trophic influence of the cells in the anterior cornua. In peripheral neuritis we find an illustration of atrophy resulting largely from diminished functional activity of the nerve-fibres. The atrophic changes seen in the bones and soft structures of the joints in chronic lesions of the brain and spinal cord, *e.g.* hemiplegia, tabes, syringomyelia and other diseases, are striking illustrations of neuropathic atrophy, while the atrophy of the hair and skin in some cases of alopecia and the white atrophic patches seen in morphœa are illustrations in the cutaneous system. As examples of atrophy from disuse or diminished functional activity, we find the muscles of a limb after amputation, the disused part of the alimentary canal in cases of organic stricture, or after colectomy, the muscles in the neighbourhood of joints affected with tuberculous or other chronic disease, the atrophied left ventricle in advanced cases of mitral stenosis, and the corpora quadrigemina after excision of the eye.

In other instances we find atrophy due to pressure and interference with nutrition, as in chronic venous congestion of the liver and other glandular organs. It is also important to indicate the part played by *inflammation* in the development of atrophy in different organs and tissues. Thus we find atrophy of the liver-cells in cirrhosis and acute yellow atrophy; in the kidney in chronic interstitial nephritis; in the optic nerve, retina, and choroid as the result of acute or chronic changes; and in the nasal mucosa in atrophic rhinitis (ozœna). A few cases of local atrophy are met with which do not admit of a satisfactory explanation, *e.g.* atrophy of the thyroid gland in myxœdema and the rarer atrophy of the suprarenal in a few recorded cases of Addison's disease.

While atrophy from disuse or diminished functional activity is common, a similar condition occasionally follows excessive stimulation with increased functional activity. Cases of this nature are seen in the nervous, muscular, and glandular systems, and as examples may be mentioned atrophy with degenerative changes in the cerebral cortex, atrophy of the biceps, deltoid, or other muscles, and a similar condition in the sexual organs. While the existence of a causal relationship between the over-stimulation and the atrophy is in most instances undoubted, the exact etiology is not so clear. It is probable that faulty chemistry in the tissues plays a very important part, the excessive stimulation leading to the imperfect removal of waste products, the accumulation of which, if maintained, leads to a permanent alteration in structure and function.

Differential Diagnosis.—This in most cases is readily arrived at by the ordinary methods of examination available; in the case of the internal organs it may or may not be possible. It is, however, of greater importance to determine the significance and cause of the condition. Cases dependent on structural alterations in the nerve centres or fibres are more pronounced than those of a simpler origin. The causal factors referred to under local atrophy should be each in turn carefully investigated.

In cases of local muscular atrophy great assistance in diagnosis may be derived from the use of the galvanic current. The presence and degree of the reactions of degeneration is a valuable gauge of the cause, and an important guide in prognosis of these cases.

Principles of Treatment.—From a study of the various causes referred to in the production of general and local atrophy, it will be seen that no general rules can be formulated for treatment. The cause of either must in each case be removed if possible, and the nutrition of the affected parts maintained by every possible means. This latter is effected by the use of measures which improve the state of the circulation, general or local, and by means which improve the general and local neuro-muscular tone. Among these, in cases of local atrophy, are massage, various physical exercises, and electricity; and here, as in cases of general atrophy, the judicious use of the natural remedies of light, air, and water (hydropathy) may prove of the greatest service. The value of these measures on local and general metabolism, and their possible influence on the higher centres in the brain, must always be borne in mind.

Atrophy, Infantile.

SYNONYMS.—MARASMUS, MALNUTRITION, ATHREPSIA.

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DEFINITION.—"Infantile atrophy" is the name applied to a condition of extreme wasting, associated with gastro-intestinal disturbance, seen in infants under a year or eighteen months. The course of the disease is usually chronic and for the most part ends fatally. Both tuberculosis and hereditary syphilis give rise to malnutrition, and this may become extreme before death, but neither of these diseases as causes of infantile atrophy need be considered here. As a matter of fact, the term "infantile atrophy" is usually reserved for those cases which are dependent upon the failure of the digestive organs to make good the wants of the body incident to life, while at the same time there is an absence of any organic disease, except perhaps a catarrhal or wasted condition of the alimentary tract. It must be borne in mind that between chronic dyspepsia with malnutrition and infantile atrophy there is no hard and fast line.

ETIOLOGY.—The chief factors in the production of infantile atrophy are: (1) *Constitutional weakness or want of vitality.* Infants born prematurely are apt to become atrophic, or the parents are weakly, and the infant inherits a tendency to dyspepsia. Some serious illness, such as an acute attack of summer diarrhœa or pneumonia, may leave the infant prostrate; it never recovers its full powers of digestion and gradually passes into an atrophic state.

(2) *Feeding with improper food.*—Infants fed at the breast of a healthy mother never suffer from atrophy, at least, if the surroundings are favourable, but it may happen that an infant fed at the breast suffers from semi-starvation, if the breast milk is poor in quality and deficient in quantity. It is the artificially fed infant that becomes atrophic, especially those who are given stale milk, boiled bread, or other starchy foods; and when once a condition of serious dyspepsia has been set up and the infant's digestion seriously weakened, it continues to waste in spite of more wholesome food being supplied. This is especially true of infants under three or four months of age.

(3) *Unhealthy surroundings.*—The infant's digestion is undoubtedly influenced by its surroundings. The infant born in a back street breathes the foul air of crowded and badly ventilated dwellings, it is exposed to cold, it lies all day and night in contact with damp and filthy napkins, it is a stranger to fresh air, and sunlight, and too often to all the numerous little attentions and care which are such important factors in preserving health. Warmth, cleanliness, fresh air, sunlight, beyond doubt, greatly influence the digestive process.

In the majority of cases of infantile atrophy, bad food and unhealthy surroundings are the chief means in producing the disease. It is among the infants born in the crowded tenements of a great city that atrophy most abounds. It is among the inhabitants of the lowest quarters of our cities that illegitimacy and early and improvident marriages are commonest, and the infants born are not only constitutionally weak, but the parents are poverty-stricken and ignorant, and the infant is only too likely to be reared and fed under the worst possible conditions. It may happen, however, that as long as the infant is fed at the breast of its mother it thrives fairly well, and is able to resist the evil influences of its surroundings. But before long, perhaps, the mother has to go to work, or for some other reason begins to give it artificial food, and at once its troubles begin. It suffers from dyspepsia and wasting, various foods are tried, boiled bread, condensed milk, dried milk foods, but all to no purpose, it loses weight with startling rapidity, and when it is reduced to little else than skin and bone, it is brought as a last resort to the out-patient room of a hospital. In other cases there may be a history of the most troublesome indigestion from the first, or the infant was doing well till it was attacked by diarrhœa, and having passed through the acute stage began to rapidly waste instead of convalescing.

Symptoms.—The early symptoms are those in connection with indigestion; of these, sometimes gastric, at other times intestinal are the most prominent; there is frequent vomiting, or colic with distension of the abdomen, or the stools are liquid and frequent. The infant is restless and in constant discomfort, and wears out the patience of its friends. The most characteristic and constant symptom is a progressive loss of weight. In a few cases there is gradual loss of flesh without any marked symptoms of dyspepsia. When the disease is well marked, the infant's appearance is very characteristic; the face is sunken from the absence of subcutaneous fat, the skin hangs in folds and wrinkles, giving it a look of old age quite foreign to an infant. The ribs and spinal prominences stand out plainly when the infant is seen stripped, the rounded abdomen contrasts strangely with the narrow chest, and the diaphragm is seen during inspiration to draw in the lower part of the chest wall. The extremities are wasted, the skin hanging loosely on the bones, the feet and hands are cold and of a purplish colour from sluggish circulation of blood. The skin has an earthy tint, and the abdomen emits a sickly sour-milk smell. In some cases, especially those that have suffered much from diarrhœa, the extremities are œdematous, the backs of the feet and hands readily pitting on pressure. Less frequently the face is œdematous; when it occurs it is a sign of bad import. In these cases there is no true nephritis, the œdema being due to anæmia and a failing circulation of blood. Excoriation and erythema of the buttocks are common, and so also are boils and bed-sores on the sacrum and heels. The temperature taken in the rectum is subnormal, often being as low as 95° or 96° F.; an elevated temperature suggests a complication such as septic pneumonia or tuberculosis. "Thrush" is also very common. The stools vary in number and consistency; in some cases they are large in amount and consist of little else than undigested food, being putty-like in appearance and containing little bile. They may be frequent and loose, consisting of stinking and decomposing food, or they are brown and slimy from the presence of mucus and altered bile. In milder cases they are perhaps green and curdy. The infant's appetite is usually ravenous, it cries at the sight of food, it is appeased for a short time by the bottle, but soon the whining cry breaks out again. In the later stages it is more apathetic, and cares less about food of any sort.

The course of the disease may be chronic, and there may be slight improvements, but for the most part the course is from bad to worse. Infants that were plump and full-sized at birth will perhaps fall away so that at three or four months they will weigh only five or six pounds. At any time the atrophic infant may be carried off by some intercurrent disease. A hot July or August at once sends up the mortality of this class, death following quickly upon an attack of diarrhœa. Measles and pneumonia are responsible for some deaths. Death occurs suddenly at times, the infant perhaps being found dead in bed. Other cases gradually get into a more and more apathetic state, they refuse their food, finally coma supervenes, the infant lying insensible with contracted pupils and Cheyne-Stokes respiration. Muscular twitchings may precede the fatal event.

MORBID ANATOMY.—An examination of the body of an infant dying of infantile atrophy will show that all subcutaneous fat has disappeared, the muscles are wasted and shrunken, and all the organs show a loss of weight, except perhaps the liver, which in some instances is large and fatty. Hypostatic pneumonia is not uncommon. In rare cases, thrombosis of the renal veins, pulmonary arteries, and longitudinal sinus may be found. The chief interest is associated with the condition of the stomach and intestines. The walls of the stomach and intestines are thin and translucent, and the solitary glands and Peyer's patches have for the most part disappeared. The surface of the mucous membrane is slimy and pale in colour, though in some cases the mucous membrane of the lower end of the ileum and colon is thickened and congested. Microscopic examination of the walls of the stomach and intestine usually shows various stages of chronic catarrh, and in the later stages atrophy of the tubular glands and villi; but some observers have failed to find any change. From these findings it would appear that the condition above described is essentially a chronic gastro-intestinal catarrh passing into a later stage of atrophy of the glandular apparatus and villi. It is, however, right to say that some competent authorities dissent from this view, but believe that the atrophy of the intestines is only part and parcel of the general wasting and not the primary cause, and that these cases of infantile atrophy are due to constitutional weakness and want of vitality.

DIAGNOSIS.—As already remarked, wasting is only a question of degree, the term "infantile atrophy" or "marasmus" being applied to the extreme form of emaciation, in which the infant may linger for weeks or more. In the most hopeless forms the emaciation is extreme, and the stools contain large quantities of decomposing food which have come through the alimentary canal without being digested. The cases attended by frequent vomiting are usually the most difficult to treat and the most rapidly fatal. The onset of acute diarrhœa is of bad augury. It is often difficult to distinguish between atrophy and general tuberculosis. An intermittent temperature, consonant râles in the lungs, induration felt through the abdominal walls, would necessarily suggest tuberculosis. Sometimes diagnosis is impossible during life, but the chances are always in favour of atrophy, especially during the first six or eight months. The diagnosis between simple atrophy and hereditary syphilis with wasting is not always easy. It must be well borne in mind that excoriations and erythema corresponding to the area covered by the napkin are no evidence of syphilis, but of course such may occur in a syphilitic infant. The tendency is to diagnose syphilis in atrophic infants without sufficient reason. Chronic coryza, anæmia with an enlarged spleen, would necessarily strongly suggest syphilis. Coryza with excoriation at the angles of the mouth, plaques or patches of erythema on the face, trunk or limbs, as also epiphysitis, are diagnostic.

TREATMENT.—The most important indications consist in protection from cold and loss of heat, supplying an unlimited amount of fresh air, and a food which the infant is able to digest. The wasted infant should be wrapped in cotton-wool and dressed in woollen clothes, which must be loose and easily removed, so as to avoid fatigue in dressing and undressing. Artificial heat should be supplied by means of hot-water bottles placed in the cot or carriage. Its bed-room or hospital ward must be large and airy, and the air frequently changed. Atrophic infants do not thrive in crowded hospital wards, and are exceedingly likely to die of septic pneumonia. It should spend as much time in the open air as the weather permits. If the infant is under three or four months of age, a wet nurse offers the best chance for it, but in this country it is seldom possible to obtain a suitable one. Fresh asses' milk is worth a trial, but this is not often to be had. Of other foods, whey prepared from cow's milk by means of Benger's essence of rennet is certainly one of the best. The curd should be thoroughly broken up, and the whey strained through muslin or a fine sieve and sterilised. Whey prepared in this way contains on an average—proteids ·8, fat 1·75 to 2, lactose 4·5 per cent. The quantity taken must be carefully regulated to the infant's powers of digestion. Atrophic infants do not digest either proteids or fat well, and the percentage of each of these must be kept low. Sugar of milk may be added to the whey in the proportion of two or three drachms to the pint. Small quantities of raw beef juice may also be added cautiously, in some cases with advantage. If cream is added, vomiting or diarrhœa is very likely to follow. If the infant improves, small quantities of milk, say one-sixth to one-third, may be added to the sterilised whey, but even this small quantity requires watching; weak peptonised milk is of use chiefly in the cases in which there is much vomiting. When there is much looseness of bowels, veal tea and thin arrowroot may be given for a while instead of milk preparations, or the whey may be diluted with an equal quantity of barley water. The medicinal treatment is very largely a treatment of symptoms. The inunction of sweet oil appears to have some good effects; a few drops daily, internally, of cod-liver oil is useful in those cases in which constipation is a marked feature, and there is no concomitant gastro-intestinal disturbance. Carbonate of magnesia in laxative doses—2-3 grains or more three times daily—with spirits of chloroform 2 minims, and peppermint water to a drachm, may be given when there is flatulence and decomposing food in the stools; Dover's powder, ¼-½ grain once or twice a day, when there is colic and diarrhœa; the dose may be increased if carefully watched, but opium in all forms is contra-indicated in the later stages when there is a tendency to drowsiness. Carbonate of bismuth in full doses, 2½-5 grains three or four times daily, may be tried to check vomiting and diarrhœa. Alcohol in the form of brandy or mist. vini Gallici is sooner or later necessary.

MEDICO-LEGAL.—It often happens that a coroner's inquest is held to inquire into the cause of death in the case of an infant, which it is alleged has been starved or grossly neglected by its mother or caretaker. At the autopsy it is found to be terribly emaciated, weighing, perhaps, only half the average weight for its age; there is no tuberculosis, and a general absence of any sign of disease which would be likely to cause death. It is not unnatural that the opinion should be formed that death was due to actual starvation, or at any rate suitable food was withheld, and the evidence given accordingly. There is not the least doubt that caution here is necessary; the absence of all traces of food in the stomach and intestines, and also of fæces, would justify the assertion that no food had been given during the last day or two of life, but the emaciated condition and absence of any trace of disease would not of themselves justify the assertion that the infant had been actually starved by its caretaker, or even grossly neglected. Emaciation of an extreme character takes place in some cases, although food in the shape of diluted milk, condensed milk, and boiled bread have been given in abundance, and there is no means at the autopsy of distinguishing between the cases in which the infant has failed to digest and assimilate the food given, and those from which food has been wilfully withheld entirely or in part. Histological examination of the stomach and intestine would be laborious and difficult, and the results inconclusive, as the absence of catarrhal changes and secondary atrophy would not necessarily mean that the infant had been starved. It is only safe to say that the appearances are *consistent with* starvation and neglect; it is unsafe, in the absence of evidence observed during life, and on post-mortem evidence only, to assert wilful starvation. The history of the case, its surroundings during life, the character of the food given, must be taken into account. If the infant is puny and weakly from birth, suffers much from dyspepsia, and dies in an emaciated condition at a few months old, or if there is a history of some acute disease, such as summer diarrhœa preceding its beginning to waste, there is no *prima facie* evidence that the infant has been neglected or badly cared for. Such cases occur in hospitals and under circumstances where neglect cannot be alleged. On the other hand, if the infant was healthy at birth, and remained so for some months, and only began to waste when five or six months old, gradually becoming reduced to a skeleton, there would be much more reason to suspect neglect or something worse. The older the infant, the less likely is it to die of atrophy unless badly fed and ill-cared-for. The presence of tubercular disease, though consistent with neglect, would probably be held by the court to account for the wasting, without alleging starvation or gross negligence.

LITERATURE.—W. SOLTAU FENWICK. *Disorders of Digestion in Infancy and Childhood*, chap. v.—EMMETT HOLT. *Diseases of Infancy and Childhood*, chap. v.

Auditory Nerve and Labyrinth.

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PHYSIOLOGY.—The eighth or auditory nerve—subserving two functions, audition and equilibration—is in two halves, the cochlear and the vestibular, each starting in a different part of the labyrinth and ending in a different part of the central nervous system, the former in the auditory cortical centre in the superior temporo-sphenoidal convolution, the latter in the middle lobe of the cerebellum.

The ACOUSTIC or COCHLEAR FIBRES, commencing in the auditory hair-cells of Corti's organ in the duct of the cochlea, pass through the spiral ganglion and join in the deepest part of the internal auditory meatus to form the cochlear division of the eighth nerve. It passes along the meatus in company with the vestibular nerve, the facial, and the pars intermedia of Wrisberg, to enter the hind brain at the junction of the pons and medulla, just dorsal to the restiform body. It here gives some fibres to the cells of the accessory nucleus, and others to the ventral or anterior auditory one. From these the fibres cross in great part to the opposite side, some doing so through the striæ acousticæ on the floor of the fourth ventricle, others more ventrally in the trapezoid body of the pons. These two groups unite in the lateral fillet, pass through the internal corpus geniculatum and posterior part of the internal capsule, and turn outwards and downwards into the corona radiata of the superior temporo-sphenoidal convolution to that part of the cortex localised as the auditory centre. Strands go from the auditory nuclei to both superior olives and posterior corpora quadrigemina. There is no doubt that each ear is connected with the auditory cortical centres of both sides, as is evidenced by the fact that although the centre for speech is situated in the left auditory centre, speech is heard by the left ear alone as well as the right, and cochlear fibres apparently pass directly from the auditory nucleus to the posterior corpus quadrigeminum of the same side (Ferrier, Turner, Obersteiner).

THE VESTIBULAR NERVE.—The fibres starting from the hair-cells of the cristæ and maculæ acousticæ of the semicircular canals, utricle and saccule, after passing through local ganglia unite in the internal auditory meatus to form the vestibular nerve. This runs with the cochlear nerve as far as the restiform body, on the ventral aspect of which it enters the hind brain to reach the external (and internal) nucleus of Deiters. Deiters' nucleus receives fibres from the roof-nucleus of the cerebellum, which in turn is connected with the cortex of the middle lobe. This is the main centre for the co-ordination of the movement for the maintenance of equilibrium, with which consequently the semicircular canals are in very close relation. The reader is referred to the articles "Brain," "Cerebellum," and "Equilibrium" for a consideration of the mechanism of equilibrium. It may, however, be noted that afferent fibres for muscular sense impressions run to the cerebellum in the posterior columns, the "comma" bundle (in Burdach's column) and the direct cerebellar tract, while efferent fibres go from Deiters' nucleus to the anterior horns of the cord through the anterior and antero-lateral columns, thus directly affecting the muscles. There are also fibres of communication between this nucleus and those of the motor nerves for the muscles of both eyes (Bruce).

AUDITORY FUNCTIONS. — *Air- and Bone-Conduction — Weber's and Rinne's Tests.*—We may accept the principle that sounds are normally perceived when the vibrations on which their existence as such depends reach the hair-cells of Corti's organ in the cochlea. This conveyance generally takes place through the structures of the tympanum, namely, the tympanic membrane, the chain of malleus, incus, and stapes, the last communicating its movements to the perilymph, through this to the elastic walls of the membranous labyrinth, and to the scala vestibuli of the cochlea, the endolymph of which is set into wave motion, identical in character with the compound wave proper to the sound, and the hair-cells are thereby stimulated. Such is the course of events when sounds are conveyed through the air, and it is known otologically as "air-conduction." On the other hand, vibrations may reach the endolymph, the hair-cells, or the nerve directly through the bones, as when a tuning-fork or other vibrating body is placed in contact with the skull. This is "bone-conduction," and is not impaired when the tympanic conducting apparatus is *alone* diseased, but only when the "perceptive" auditory apparatus in its peripheral cochlear cells, its trunk, nuclei, central continuation or cortical centre is organically—or functionally—affected.

Weber's and Rinne's Tests.—Any condition which obstructs the entrance of air-conducted sound waves from without acts at the same time as a hindrance to the escape of bone-conducted waves from within.

If a vibrating tuning-fork is placed on the vertex it is in the normal person heard equally in both ears or apparently in the middle of the head. If, however, one ear be stopped up with the finger, a mass of cerumen, an over-tense tympanic membrane or ossicular chain, the tone is heard louder and exclusively in the obstructed ear. This is *Weber's test*, which is said to be "positive" when the fork is heard louder in the deafer ear—indicating obstruction; "negative" when louder in the better ear—indicating nerve-deafness in the worse. In pure obstructive deafness the tuning-fork is heard by the patient (on his mastoid) after it is no longer heard by the normal observer. This is "*increased bone-conduction*" test. In pure nerve-deafness there is "*diminished bone-conduction.*"

In most instances increase of bone-conduction indicates presence of obstructive disease and absence of nerve affection. Steinbrügge attributes increased bone-conduction to heightened sensibility of the auditory nerve quite as much as to mechanical obstruction, and that its occurrence is not to be taken as diagnostic of the latter condition. The occurrence of hyperacusis is undoubted when it applies to air- as well as bone-conducted sounds, but the coincidence of heightened excitability of the auditory nerve along with an eclipsing affection of the apparatus conducting external sounds must be rare, though obviously not impossible. The "obstruction" origin of increased bone-conduction is certainly the most intelligible and the most frequently applicable explanation.

The normal tympanic apparatus is a better conductor of sounds—at all events for those of low or medium pitch—than the cranial bones, and when a vibrating tuning-fork of, say, 256 d. vs. (C′ = 256 d. vs.) placed in contact with the mastoid region of the skull dies down till it is no longer heard, it is still audible for some seconds if held opposite the meatus. This was observed by Rinne, and is known as the normal or "*positive Rinne*" ("Rinne + ").

Any condition hampering the action of the tympanic apparatus (inflammatory or other affections) naturally diminishes the transmission of vibration conveyed in the usual way through the air ("diminished air-conduction"), and in addition it prevents the diffusion outwards of vibration conveyed through the cranial bones. If this is at all considerable, the tuning-fork, after ceasing to be heard on the mastoid, is no longer heard when held opposite the meatus, and Rinne's test is said to give the negative result—"*negative Rinne.*" In some cases the fork is actually heard longer through the bones than through the meatus, and this exaggeration of the negative may be called "*reversed Rinne.*" In slight cases there may be merely a shortening of the time during which the fork is heard opposite the meatus after ceasing to be heard through the bones, and Rinne though positive is shortened, "*shortened positive Rinne.*"

EQUILIBRIAL OR STATIC FUNCTIONS OF THE VESTIBULAR NERVE.—*Aural Vertigo.*—It is obvious that every alteration of the position of the head must produce a disturbance in the fluid bathing the hair-cells. The viscosity of the fluid and the almost capillary narrowness of the canals must moderate this considerably, but nevertheless important afferent sensory impulses must be conveyed from these to Deiters' nucleus and the cerebellum.

Ewald attributes to the posterior part of the labyrinth an influence on the "tonus" of muscle which is exercised through the channels already described. The suppression of one or more of the semicircular canals has very slight and only transitory effect on the equilibrium, but irritation of one or more of them is accompanied by the greatest disturbance. Post-mortem examinations of deaf-mutes show that in a large percentage the semicircular canals are obliterated, and the only effect noted is that, as a class, deaf-mutes are somewhat more ungainly in their progression than other people, and enjoy immunity from giddiness when subjected to rotation.

On the whole, the canals probably co-operate with the other sensory mechanisms concerned in the maintenance of equilibrium, viz. sight, muscle-sense, and the varying pressure of the fluids in the body; they add to the delicacy of the sense of position, but are probably not essential to it. Their value is perhaps more than counterbalanced by the disturbing effect of even very slight irritations, so that changes of pressure or variations in vascularity may produce severe vertigo.

THE VARIOUS CAUSES OF VERTIGO will be found fully discussed under "Vertigo." It may, however, be here noted that Sir William Gowers attributes at least 90 per cent of all cases of definite vertigo to aural disturbance, the remainder being of gastric, arterio-sclerotic, epileptic, organic cerebral, or simply "essential" nature. Vertigo from aural disease is chiefly characterised by its definiteness (not a mere confusion, but a marked sense of movement of self or surrounding objects in a definite plane), by the comparative absence of loss of consciousness (distinguishing it from epilepsy), by its persistence in spite of closure of the eyes (unlike ocular vertigo), and above all by the *concomitant defect of hearing.* In the most pronounced apoplectiform cases the cause of the vertigo and the deafness is an effusion into the labyrinth, but in a very large number of even considerable severity the vertigo is due to irritation of the vestibular nerve-endings, produced by uncomplicated disease of the middle, or even the external, ear. Neurasthenic hyperæsthesia of the vestibular nerve may contribute to the production of vertigo. It is generally held that the rotation or sense of rotation is towards the affected ear, but to this rule the writer's experience offers many exceptions.

THE DIAGNOSIS OF DISEASE OF THE AUDITORY NERVE AND LABYRINTH. —NERVE-DEAFNESS IN GENERAL.—Disturbances of hearing and of equilibrium are the functional indications of the greatest significance, but it is the former which is the most eminently characteristic and important for localising purposes. Given dulness of hearing, we have to explain it by disease of the auditory nervous apparatus, of the conducting apparatus, or of both combined.

The tuning-fork tests above described, if well marked and intelligently interpreted, will generally guide us to a diagnosis, thus:—

Increase of bone-conduction, with negative Rinne and positive Weber, indicates an *affection of the conducting apparatus.* Decrease of bone-conduction, with positive Rinne and negative Weber, indicates an *affection of the auditory nerve mechanism.* Decrease of bone-conduction, with negative Rinne, especially if bilateral, indicates a *disease of the conducting and nervous apparatus in combination.*

It will be obvious that confusion must often arise from the interference of the opposite, perhaps normal or differently affected ear and from the coincidence of disease of the conducting apparatus. It is impossible to give rules to meet every eventuality, but the following statements may be fairly depended on:—

"Positive Rinne" may be accepted as excluding any considerable degree of obstructive disease, and any such degree of deafness must in its presence be due to a nerve affection.

"Negative Weber" in unilateral deafness indicates nerve-deafness (even if accompanied by "negative Rinne").

The following cautions will also aid:—

"Negative Rinne" is common in unilateral nerve-deafness, the bone-conduction being apparently good because the vibrations are conveyed by the bone to the opposite ear. (It has to be checked by Weber's test.)

In cases of obstructive—catarrhal—deafness in one ear, with sympathetic nerve-deafness (not an uncommon occurrence) in the other, the increased bone-conduction in the former makes the negative Rinne in the latter all the more pronounced. (It can only be checked by the comparative decrease—or absence of increase—of bone-conduction, by the loss of hearing for high rather than low tones, and by the sequence of events in the case.)

Further evidence that the condition is an affection of the internal auditory apparatus may be derived from the following symptoms:—

The hearing being *worse in the midst of a noise* instead of better, as in most cases of chronic bilateral affections of the middle ear.

Incorrect hearing for tones, in so far as notes may be heard a tone or more higher or lower than as heard by the normal ear, the wrong tone not being necessarily a harmonic of the true one. A single tone may therefore be heard in double (diplacusis), correctly by the healthy ear and incorrectly by the diseased one.

The loss of hearing is usually *worst for high tones* (in middle-ear disease it is worst for the lower ones), but there may be actual *gaps* in the range of hearing.

The *subjective noises*—tinnitus aurium—produced by disease of the internal auditory-nerve apparatus vary so as to have very little diagnostic value. A noise checked by compression of the vertebral arteries in the suboccipital hollows is strongly characteristic of labyrinthine congestion. It is thought that sounds of labyrinthine origin have a more definite musical pitch than those due to middle-ear disease. Tinnitus without dulness of hearing is attributable to neurasthenic irritability of the auditory centres, and is often associated with abnormal sensibility to sound (hyperacusis). Elaborated tinnitus, taking the form of voices uttering spoken words, is a cerebral or mental affection. It is an *auditory illusion* if it is merely an erroneous interpretation of a simple tinnitus produced by a detectable disease of the middle or internal ear, a *delusion* or *hallucination* if no such exciting cause is present.

Vertigo is more characteristic of internal than middle-ear disease, especially in the acute stages.

Fatigability of the Auditory Nerve.—As in the case of other sensory nerves, stimulation of the auditory nerve is followed after a time by fatigue and insensibility. This passes off after a very short rest, and the hearing power again returns. If a vibrating tuning-fork is held opposite the ear till no longer heard, is then removed for a second, and replaced in its former position it is, as a rule, again heard for a longer or shorter period. With a long-sounding fork this may be repeated more than once. The fork to be used must be tested on the normal subject, so that it may be noted *how often* (after the maximum stroke) *it is normally lost and reheard*, and, if possible, how long each time. In cases of "increased fatigability" of the auditory nerve or centres this occurs more often than normal. Gradenigo has found this fatigability to be most pronounced in disease of the auditory-nerve trunk. He tests it further by observing the degree to which the hearing power is reduced by the sudden exposure to a loud sound. It sometimes happens in the course of the ordinary tests for bone-conduction that a fork reheard on the mastoid after ceasing to be heard at the meatus is also reheard at the meatus after ceasing to be heard on the mastoid. These contradictory results are obviously to be explained by abnormally rapid fatigue of the nerve.

Inflation of the tympanum usually increases the deafness in disease of the internal ear.

LOCALISATION OF THE SEAT OF THE LESION PRODUCING "NERVE-DEAFNESS." We are led in this chiefly by the following points:—

The part of the range of hearing chiefly affected.

The presence or absence of symptoms indicating simultaneous affections of—

1. The equilibrial portion of the labyrinth.
2. Other cranial nerves.
3. Other portions of the central nervous system.

The *part of the range of audition affected* is tested by means of tuning-forks for the lower and middle tones, and by Galton's whistle or Koenig's rods for the highest ones. Loss of hearing for the tones of the highest pitch (lowering of the upper limit of audition) is characteristic of an affection of the labyrinth, and is indeed our chief localising feature. On the other hand, if with the general tuning-fork indications of "nerve-deafness" the hearing for the highest tones is comparatively preserved, but for lower ones diminished or lost, we have to deal more probably with disease of the deeper parts of the auditory tract, either organic or (not uncommonly) functional or hysterical.

The simultaneous occurrence of violent vertigo, along with considerable nerve-deafness, would indicate that the *labyrinth was affected in both the cochlear and ampullary sections*, more especially if there is at the same time absence of signs of involvement of the cranial nerves or the central nervous system. This is especially characteristic in hæmorrhage into the labyrinth.

Simultaneous affections of other cranial nerves.—These localise the affections as in the following situations:—

(*a*) Nerve-deafness with facial paralysis (seventh) alone, without chronic aural suppuration, localises the internal auditory meatus; with chronic aural suppuration, the bony labyrinth.

(*b*) With paralysis of one vocal cord and half of the soft palate, rapid pulse (vago-accessory nerve), disease behind the apex of the petrous bone—often a pachymeningitis, sometimes a new growth.

(*c*) With convergent strabismus of the eye of the same side (sixth nerve), a lesion on the under surface of the pons, generally meningitic, involving the auditory nerve and often extending into, or from, the labyrinth.

(*d*) With conjugate deviation of both eyes away from the affected side (sixth nerve of affected side, and internal rectus fibres of opposite third nerve) we could expect a lesion of the auditory nuclei in the medulla.

Symptoms of disease of other portions of the central nervous system:—

(*a*) Locomotor ataxy indicates tabetic auditory neuritis.

(*b*) Cerebellar symptoms (reeling, vomiting, headache, optic neuritis, paresis of the arm of the same side, etc.) probably indicate compression of the auditory nerve by a cerebellar tumour.

(*c*) Bulbar symptoms (paresis of tongue and lips), a lesion of the auditory nuclei in the medulla.

(*d*) "Quadrigeminal" symptoms (blindness or optic neuritis, incoördination of the movements of the eyes, unsteadiness of gait, etc.), a lesion of the auditory tract in the tegmentum—lateral fillet—as from pressure of a pineal or quadrigeminal tumour.

(*e*) Hemianæsthesia and hemianopsia on the same side as the loss of hearing suggest hysteria or a lesion of the posterior part of the internal capsule.

(*f*) "Word-deafness" indicates a lesion of the upper left temporo-sphenoidal convolution (in left-handed patients the right one).

PATHOLOGICAL NATURE OF THE LESIONS.—We are guided in this very much by the mode of onset, thus:—

A sudden onset is characteristic of hæmorrhage, congestion, or anæmia, whether traumatic or idiopathic, embolism or hysteria.

A rapid onset with fever is seen in the various forms of the acute labyrinthitis, *without fever* in such rapid effusions as occur in syphilitic affections of the labyrinth.

A gradual onset suggests the slower inflammatory or degenerative changes resulting from habitual exposure to noises, sclerosis of the middle ear, toxic agencies, plethora, anæmia, degenerations of the central nervous system, tabes dorsalis, new growths and old age.

A congenital defect indicates imperfect development or ante-natal disease.

SPECIAL DISEASES OF THE LABYRINTH AND AUDITORY NERVE-APPARATUS

In the following account, such pathological details as are available are treated with the utmost brevity to allow space for points of clinical and practical importance.

Anæmia of the Labyrinth and Auditory Nerve generally arises from causes such as rapid and considerable loss of blood, continuous defective nutrition, or rarely from local causes, such as occlusion of the auditory artery by pressure of a tumour, embolism, or thrombosis. Small hæmorrhages have been found in the labyrinth in cases of pernicious and simple anæmia.

It is recognised as a "nerve-deafness" associated with pallor, faintness, and the other general signs of anæmia, with humming, often pulsating tinnitus. The symptoms are relieved by food, by rest in the recumbent posture, and, for the time, by inhalation of a small quantity of nitrite of amyl. The most characteristic instances occur in hospital practice in the persons of poor widows. *Treatment* consists in good nourishment, judicious use of alcoholic stimulants, iron, arsenic, chloride of ammonium, hæmoglobin preparations, and increasing doses of strychnine or nux vomica.

Congestion of the Labyrinth and Auditory Centres occurs alone, but is often the accompaniment of congestive affections of the middle ear or of the encephalon, whether arising from inflammatory or neuro-vascular causes. We frequently find this last factor in action during the climacteric, or other periods of nervous disturbance. It also arises from conditions increasing the arterial tension (chronic interstitial nephritis, gout, etc.) or impeding the venous return (goitre, heart disease, aneurysm, etc.).

The characteristic features are in general "nerve-deafness," with evidences of plethora, such as florid complexion, headache, rushing or pulsating noises in the head, impaired sleep, attacks of vertigo, or loss of blood. The symptoms are relieved by purgatives, and are made worse by lying down, hearty eating, alcoholic stimulants, mental excitement, or for a time by the inhalation of nitrite of amyl. In arterial congestion the tinnitus is usually pulsating, and it is checked by compression of the vertebral arteries by means of the finger and thumb of one hand in the hollows behind, and slightly below the mastoid processes. In venous congestion there is more of a rushing noise, and pressure on the vertebrals has a less marked effect.

Treatment consists in the removal, if possible, of such causes as have been mentioned. In general, the diet will need regulation or reduction; aperient drugs or waters will have to be ordered. The most valuable remedies are the bromides, and in many cases, especially the climacteric, a combination of bromide of potassium with the compound decoction of aloes will be found reasonably palatable and therapeutically of the utmost value. In cases of arterial tension, small doses of calomel, $\frac{1}{6}$ to $\frac{1}{2}$ a grain once or twice a day, with a milk and fish diet, are very efficacious. Relief sometimes follows depletion of the turbinated bodies. Pressure may be exerted on the vertebral arteries by small conical pads in the suboccipital hollows above mentioned, kept in their place by means of a strap round the forehead. Pilocarpine is sometimes beneficial in these cases, but in the anæmic ones it is to be rigorously tabooed.

Vaso-motor Oscillations in the Auditory Circulation.—In some cases we find auditory and equilibrial disturbances in the same individual, presenting at one time congestive, at another anæmic characters, which we may attribute to vaso-motor instability from whatever cause arising. Among the most frequent causes are abuse of tobacco, sexual excitement and nervous exhaustion. The removal of the cause is the primary indication. This may be accompanied by the administration of small doses of nerve tonics, such as arsenic, strychnine, ignatia, and, perhaps best of all, camphor, which may be administered in doses of five to ten minims of the strong (Rubini's) solution, in half a wine-glassful of milk three or four times daily. Bromides must be prescribed with caution. The bromide of arsenic (Clemens' solution) with strychnine and hydrobromic acid combine well. The application of the continuous current to the "sympathetic" in the neck has been strongly recommended.

Hæmorrhage into the Labyrinth.—This is the lesion typical of "Méniere's disease." It may occur in the course of any of the general diseases which lead to hæmorrhages, such as leucocythæmia, purpura, whooping-cough, and simple or pernicious anæmia, but also, apart from any of these, especially on exposure to cold either after working in a hot place, or when the menses are overdue. Gout and other diseases causing increased arterial tension favour its occurrence. Not being a fatal disease, the *post-mortem* observations are extremely few. In the typical case the hæmorrhage may be presumed to plough up the endolymphatic elements of the cochlea and semicircular canals.

Symptoms.—The patient is seized with a violent confusion and giddiness, so great as to upset his equilibrium, and make him drop down on the floor. He breaks out into a cold sweat and generally vomits. With this he experiences loud tinnitus in one ear, which he then or only later discovers to be quite deaf. On a hurried glance the case most resembles a "severe bilious attack," for which it is very frequently mistaken, until the patient discovers the deafness of one ear, and the signs of nerve-deafness are detected (diminished or extinguished bone-conduction, negative "Weber," positive or most usually the paradoxical negative "Rinne," the loss of hearing, including tones of the highest pitch, etc.). The hearing is rarely recovered. Similar attacks, but of much slighter severity, may arise from sudden congestion or anæmia or from minute hæmorrhages. These more resemble epileptic seizures, but are to be distinguished by the aural symptoms and by the absence of loss of consciousness. For some days, or even a number of weeks, the nausea and vertigo may necessitate the recumbent posture, and the patient may long remain subject to sudden attacks of vertigo, causing him to fall, or in any case to catch hold of some support.

Treatment.—The first point in the treatment is to diminish the violence of the nervous shock by placing the patient in the recumbent position in a quiet, darkened room, and administering full doses of the bromide of potassium. Ice may be applied to the head while the body and feet are kept warm. When the shock has passed off an endeavour should be made to induce absorption of the effusion by the administration of pilocarpin, and of iodide of potassium. The former may be given hypodermically in doses increasing from $\frac{1}{12}$ to $\frac{1}{3}$ or even $\frac{1}{2}$ a grain daily, with precautions against chill during the perspiration and salivation which follow. The giddiness is best combated by means of quinine, and the writer's experience would lead him to consider such small doses as $\frac{1}{2}$ to 2 grains thrice daily, with 10 or 15 drops of dilute hydrobromic acid, as efficacious as the larger ones and less objectionable. The quinine appears to act on the vestibular nerve as a direct sedative, and is particularly applicable to the after period, when the patient remains long subject to sudden vertiginous attacks on the slightest provocation. Mackenzie considers rise of pulse-tension the most frequent excitant, and recommends the taking of a dose of calomel on the occurrence of the slightest sensation of giddiness.

Inflammation of the Labyrinth may be acute or chronic.

Acute Labyrinthitis.—The most common forms arise from microbic invasion in the course of infectious diseases, such as scarlet fever, measles, diphtheria, or, in particular, mumps. Some cases arise by simultaneous invasion or by extension from meningitis, either simple, tubercular or epidemic, others—primary simple labyrinthitis—without any such associations, and some follow injuries to the petrous bone. Again, it may occur as the result of extension from the middle ear, occasionally culminating in necrosis and possible extrusion of the labyrinthine capsule as a whole or in part.

ACUTE PRIMARY LABYRINTHITIS is characterised by the occurrence, along with high fever, of extreme vertigo, vomiting, pain in the deep parts of the ear and complete deafness. It is usually accompanied by severe mental disturbance and delirium, so as to be taken for meningitis, until the rapid subsidence of all the symptoms, with the exception of the absolute deafness, unsteadiness of gait, and tinnitus negatives the idea. The deafness is of the nerve form, answering to the typical tuning-fork tests, unless indeed it is so absolute that no tests are available or necessary.

INFECTIOUS LABYRINTHITIS in the course of a fever is characterised by the super-addition of the symptoms above mentioned to those of the existing disease, the subjective ones being of course only recognisable at the time if the patient's sensorium is sufficiently clear to allow of their being elicited. It is often only as the patient is recovering that the persistent deafness and vertigo reveal the unfortunate occurrence.

MENINGITIC LABYRINTHITIS, arising by extension of inflammation from the meninges along the sheath of the auditory nerve, is practically undetectable until the meningitis subsides and a typical cochlear nerve-deafness remains.

ACUTE TRAUMATIC LABYRINTHITIS may be diagnosed when to the symptoms of concussion or fracture of the labyrinth there are superadded those of inflammation, increased pain, giddiness, and feverish disturbance.

The treatment of acute labyrinthitis, as such, consists in giving the organ the greatest possible rest in a quiet room, with derivation to the bowels by aperients—calomel—and to the extremities mustard leaves or hot bottles, while ice is applied to the head. Depletion should be practised by means of leeches behind the angle of the jaw. When the fever subsides pilocarpin, as above described, and iodide of potassium, should be administered. Blisters behind the ear are then of value. As regards the infectious forms prophylaxis during febrile attacks is of the greatest importance, especially as regards exposure to cold. In mumps the subsidence of the parotitis is favoured by the administration of jaborandi or its alkaloid pilocarpin, and this, the chief remedy for labyrinthine effusion is thus ideally indicated.

The after results of labyrinthitis, so far as the intense deafness is concerned, can best be mitigated by instruction in lip-reading, every little remnant of hearing power being, however, cherished and encouraged by exercise in listening to shouted vowels, syllables, words or phrases, with or without the intermediation of a speaking-trumpet. When the disease occurs in children who have not yet learned to speak—as a rule under four years of age—deaf-mutism results, and children below the age of seven are apt to lose gradually the power of speech they have already acquired.

CHRONIC LABYRINTHITIS with thickening of the membranous part, deposits of fibrous calcareous and even osseous tissue may lead to the more or less complete obliteration of the cavities of the internal ear. The nervous elements undergo steady degeneration.

It may result from many causes, and may be associated or not with disease of the middle ear.

PRIMARY CHRONIC LABYRINTHITIS in its most marked form occurs as the result of acquired or inherited syphilis, but we may include under it those changes which are induced by continued exposure to noises, and by certain toxic agencies.

SYPHILITIC LABYRINTHITIS may be hereditary or acquired, and in the acquired form may occur in the secondary or tertiary stages.

The symptoms in typical cases are those of a very rapidly developed "nerve-deafness." In a day or two, or in a few hours, or even, exceptionally with all the suddenness of a hæmorrhage, the hearing of an ear may diminish even to absolute deafness. In most instances the concomitant disturbance of equilibrium is slight in comparison with the profound degree of the deafness, but in the sudden cases this is extremely pronounced. The tuning-fork tests and the relatively greater loss for high-pitched tones localise the affection. *The rapid onset* without acute pyrexial disturbance is most characteristic of its syphilitic nature, but confirmation by the discovery of other lesions must be carefully looked for.

In the hereditary form the deafness most usually appears about the period of puberty. It is generally preceded—sometimes followed—by interstitial keratitis, and it is accompanied by the peculiar peg-shaped and notched upper central incisor teeth described by Hutchinson.

In the acquired disease the most usual stage for the occurrence of nerve-deafness is the tertiary, but in rare cases it is the secondary.

The prognosis is in the secondary stage fairly good; less so in the tertiary even if treated soon after the onset of the deafness; most unfavourable if it is of long-standing.

In the hereditary form it is almost hopeless, improvement under treatment, though not unknown, being extremely exceptional.

Treatment of the acquired form in the secondary stage consists in energetic mercurial inunction or subcutaneous injection along with the administration of the hydrochloride or nitrate of pilocarpin. Of this latter drug, a 1 in 24 solution may be used, and two drops of this ($\frac{1}{12}$ grain) injected subcutaneously on the first day, an additional drop being added each successive day until the amount of 8 or 10 is reached according to the effect on the patient. After each injection the patient should remain in bed warmly covered, until the sweating has passed off. He should also have a small dish conveniently placed to catch the saliva as it dribbles from his mouth. This treatment should be carried out continuously for a fortnight under the supervision of the medical attendant, not by any means necessarily a specialist. Cardiac depression is to be combated by the administration of brandy or aromatic spirits of ammonia—which indeed favours the diaphoretic action—or in extreme cases sulphate of atropia.

In the tertiary stage it is decidely advisable—at least in otherwise fairly strong people—to give the "pilocarpin treatment" a trial. If a previous mercurial course has not been gone through, inunction should at once be practised, followed up by iodide of potassium in full doses.

In the hereditary form specific treatment is almost useless, but the observation of occasional benefit by reliable persons renders it unjustifiable to leave the recognised means untried. Mercurials have seemed to the writer to do more harm than good, pilocarpin has been equally without avail, but others have found it beneficial in a few cases. He has seen both the general condition and the hearing improved by iodide of potassium in doses of one drachm every four hours. Iodide of iron is in general the most useful remedy in doses of not less than one drachm of the syrup three or four times a day. Donovan's solution of mercury and arsenic is at times useful. Nothing is of any value without the accompaniment of good nourishment supplemented by such additions as malt extract and cod-liver oil.

LABYRINTHITIS FROM EXPOSURE TO NOISES.—The frequently observed "nerve-deafness" occurring in those habitually exposed to noises, such as boilermakers, cotton-factory weavers, artillerymen, and others, is explainable by the repeated or continual concussion to which the auditory nerve is exposed. The meagre contributions to the morbid anatomy seem to indicate that the proximate cause is a progressive inflammation and degeneration of the nervous elements in the labyrinth. The condition presents itself frequently in sportsmen and marksmen as the result of excessive shooting, the left ear being in them the more exposed, and consequently the more markedly affected.

The treatment consists in suitable change of occupation (not, for instance, as in a case of the writer's, from that of a naval gunner to that of a boilermaker!) or in the plugging of the ear during shooting, or, it may be, in the adoption of the form of powder and sporting weapon which gives the least report. This, though often beneficial, is not always so, and the degeneration once started has been known to be progressive. Improvement has, in the writer's experience, been followed in cases treated for him by Dr. Lewis Jones by means of the continuous electrical current. The negative pole is the one applied to the tragus.

A single exposure to a violent explosion has frequently produced a similar form of deafness. In one instance suction to the meatus has afforded relief apparently by drawing out the in-driven tympanic structures. As a rule the damage is in these cases permanent.

LABYRINTHINE DEAFNESS RESULTING FROM CHRONIC NEPHRITIS.—Morf's investigations would indicate that this is due to a toxic rather than an inflammatory, circulatory, or œdematous change, the deafness fluctuating with the evidences of uræmia. In the writer's experience it is usually associated with a very considerable amount of vertigo.

In general the *treatment* is that appropriate to the uræmia. In practice the symptoms, especially the vertigo, seem to yield most readily to iodide of potassium, with a "milk and fish diet."

CHRONIC LABYRINTHITIS, SECONDARY TO MIDDLE-EAR DISEASE.—We find a chronic inflammatory degeneration of the nervous elements in the internal ear in the more advanced stages of *chronic non-suppurative inflammation of the middle ear*, especially when of the sclerotic type. It may be due to extension of the arthritic disease from the stapedio-vestibular articulation—rheumatic, osteo-arthritic, gouty, syphilitic, or otherwise,—or it may be, with this arthritis, a concomitant result of a tropho-neurotic disturbance. It is to be recognised by a diminution of bone conduction succeeding to the previous increase, with the persistence of the negative Rinne, and the loss of hearing for the highest tones. There is then better hearing for middle tones than for any others.

The treatment consists primarily in the avoidance of local manipulations which do more harm than good, the counteracting of the special dyscrasia present and the maintenance or exaltation of the patient's nerve tone. We must mainly depend on the administration of strychnine (of which large doses are better borne if covered, as advised by Lake, with small doses of chloral hydrate), combined with dilute hydrobromic acid and arsenic in whatever form is best borne. Residence at an altitude is specially beneficial.

Chronic Labyrinthitis associated with Chronic Suppurative Inflammation of the Middle Ear.—This is usually of tuberculous nature, but it may arise in the course of any depressing disease, such as scarlet fever, measles, typhoid, diphtheria, or diabetes. The microbic invasion often extends through the fenestra rotunda, causing speedy disorganisation of

Corti's elements, or the bone may be primarily attacked, and spreading caries or necrosis result. Besides damage to the auditory structures, there is frequently involvement of the facial nerve, occasionally erosion of blood-vessels, infection of the meninges, sinuses, or brain. *The signs of its existence* are, in addition to the long-standing fetid suppuration from the middle ear, extreme diminution of hearing power by bone conduction, frequent disturbance of equilibrium, the presence of carious spots on the promontory, or the extrusion of fragments of diseased bone.

The treatment locally consists in the use of antiseptics, especially irrigation with perchloride or biniodide of mercury.

Constitutionally the therapeutic and hygienic measures indicated by the dyscrasia must be carried out.

VARIOUS SPECIAL FORMS OF NERVE-DEAFNESS

TABETIC AUDITORY NERVE-DEAFNESS.—This is one of the less common complications of locomotor ataxy, and may be due to a degenerative neuritis in the nerve trunk, an affection of the auditory nucleus, or a syphilitic labyrinthitis. *Neuritis*, which has been found post-mortem, would be indicated by a nerve-deafness in which the loss of hearing was greatest for tones other than the highest; *the nuclear affection* by implication of neighbouring nuclei (the sixth with conjugate deviation to the opposite side); *the labyrinth* by the intensity of the deafness, most marked for the highest tones and the rapid onset, as compared with the gradual development of the others. The primary diagnosis would, of course, depend on the co-existence of the signs of locomotor ataxy, which may be overlooked if not kept in mind, and which should therefore be sought for in every case of nerve-deafness.

The degree of deafness undergoes slight variations like the other symptoms in tabes, and there is little to recommend beyond the constitutional treatment appropriate to that disease.

SYMPATHETIC NERVE-DEAFNESS.—For some reason or other disease of one ear, whether of the conducting or the perceptive part, exercises an injurious influence on the opposite one, this showing itself as what may be well described as a sympathetic "nerve-deafness." It occurs characteristically in cases of the residua of unilateral suppurative otitis, and the hearing of the sympathetically-affected ear is frequently improved by treatment of the primarily diseased one, even though no effect is produced on the hearing of the latter.

The treatment indicated is the removal of irritating conditions in the first affected ear. This may mean the adoption of a more thorough antiseptic treatment, the removal of the ossicles, of cholesteatomatous masses, or even the opening of the mastoid cavities.

TOXIC PARALYSIS OF THE AUDITORY NERVE.—Among the most frequent causes of nerve deafness is the ingestion of *quinine, salicin*, or *salicylates*. Much more rarely it may be traced to *alcohol, tobacco*, or *lead*, and it is quite probable that *toxins absorbed* from the *intestines* may exercise a similar influence. The only evidence is the presence of the nerve-deafness, and the history, which should be inquired into in all obscure cases. Quinine deafness is generally recovered from if the drug be discontinued, and bromides or hydrobromic acid be administered. Lead is sometimes absorbed, when used as a hair-dye. Patients, when recommended to remove this cause, seldom return to report the result of following this unpalatable advice. Alcoholic deafness yields to abstinence and strychnine, but tinnitus is apt to persist long.

Nerve-Deafness in Hysteria.—Assuming that motor and sensory disturbances in hysteria are the results of self-hypnotisation, then, as the hypnotised patient to whom it is "suggested" that she is deaf *is* deaf, so the subject of hysterical deafness *cannot* hear. It is a condition totally different from shamming, though sometimes associated with it or mistaken for it.

It is sometimes unilateral and part of a hemi-anæsthesia, but more often it is "bilateral." It is clinically a nerve-deafness, and the hearing is not necessarily worst for the highest pitched tones. This peculiarity enabled the writer to suspect a hysterical origin in a very obscure case, in which his provisional diagnosis was only confirmed after several years.

The onset is *often sudden*, as the result of a mental shock, or a slight physical one, and it occasionally occurs in young girls, in whom the menses have been rather late in establishing themselves. In some instances it ingrafts itself on a pre-existing middle-ear disease.

It often eludes diagnosis, but the general aspect of the case and the presence of some of the usual stigmata of hysteria, such as "globus," hemi-anæsthesia or hemi-hyperæsthesia, diminished pharyngeal reflex, functional aphonia, and narrowing of the field of vision, may help materially.

Violent remedies are to be avoided for fear of adding "traumatic" hysterical symptoms to those already present. Hypnotism is not always easy to induce in hysterical subjects, and is open to objections. Anti-spasmodic nerve-tonics, good nourishment, general anti-hysterical regimen and encouragement, constitute the chief means of treatment, the method of Weir-Mitchell being kept in mind. In retardation of the menses the flow should be encouraged *secundum artem*.

Senile Nerve-Deafness.—After the age of sixty it is natural for the hearing-power to decrease. This change is characterised by diminished bone-conduction, positive Rinne, and marked loss of hearing for the upper tones of Galton's whistle. It is important to recognise the condition so as to avoid attempts at ineffectual and possibly injurious treatment, but it is to be remembered that it may be complicated by a curable catarrh or other affection. The chief means of help is some form of ear-trumpet, a long flexible one for conversation with one person, a parabolic one for meetings, concerts, sermons, and so forth.

Nerve-Deafness arising from Cerebral Disease.—Reference has already been made to the labyrinthine inflammation arising by extension from the inflamed meninges. The nerve-trunk may itself be infiltrated, although its more compact companion, the facial, may escape.

It is unquestionable that the auditory nerve may be injuriously affected by increase of cerebral pressure from any cause. A tumour may cause dulness of hearing, by increasing the intra-cranial tension, or by interfering with the auditory nerve-trunk or the intra-cerebral auditory tract.

The chief localising symptoms have been already indicated, and as a rule the deafness is more or less eclipsed by the other symptoms, such as blindness, headache, vomiting, disturbances of equilibrium, or of the ocular movements, paralysis of muscles of locomotion, articulation, deglutition, and so forth, which localise the site of the lesion. According to Gradenigo, the chief departure from the normal may be an increased auditory sensibility to galvanism. The sensation of sound is not excited in the normal nerve by a current (closing cathode) of much less than 10 milliampères, but when the sensibility is increased much weaker currents may suffice.

Traumatic Nerve-Deafness.—*Injuries to the labyrinth* may result directly, as from the introduction of pointed bodies through the meatus, or indirectly, as from blows or falls on the head. These may, in slighter cases, induce a concussion of the labyrinth, which may completely subside, but in severer cases, with symptoms of fracture of the base of the skull, the osseous capsule of the labyrinth is usually involved, with irreparable destruction of its contents. It is apt to be followed by suppurative inflammation.

The chief indication for treatment is, in the first instance, the adoption of antiseptic measures for the prevention of infection from without. This should take the form of gentle syringing of the meatus with a 1 in 3000 solution of biniodide or bichloride of mercury, or with the "Lister" strong solution, the cleansing of the auricle, the insertion of iodoform gauze in the meatus, and the covering of the ear with an antiseptic dressing. Complete rest and quiet, with cold to the head, are advisable. The restoration of hearing is scarcely to be considered, but the resulting vertigo in these traumatic cases is extremely amenable to treatment with small doses of the solution of perchloride of mercury (min. xv. to xxx., thrice daily).

NEW GROWTHS.—The auditory nerve may be the primary seat of new growths, such as fibroma, neuroma, myxoma, psammoma, and sarcoma, but it is also often involved in growths extending from neighbouring parts.

Non-malignant tumours affecting the auditory nerve primarily are almost beyond the range of diagnosis, but may be suspected when a nerve-deafness with vertiginous attacks persists, and slowly increases, especially if it does not involve particularly the highest portion of the range of audition. Gradual extension to other nerves, such as the facial, the vago-accessory, hypo-glossal or sixth, in spite of anti-syphilitic treatment, is the practical summary of the indications. Interference with the salivary secretion of the same side results from the involvement of the pars intermedia of Wrisberg, now believed to be the origin of the chorda tympani.

Malignant Growths are more rapid in their aggression, and usually show themselves at some time by protruding through the petrous bone into the tympanum or meatus, simulating granulations, or into the mastoid region to be mistaken for an inflammatory affection of that part. Thus mastoid operations have been performed in cases of sarcoma. The nature of the disease is sometimes identified on microscopical examination of a fragment.

CONGENITAL NERVE-DEAFNESS.—In proportion to the frequency of malformation of the external and middle ear, defective development of the internal ear is rare. More often the congenital abnormalities are the result of ante-natal disease, as indicated by the identity of the conditions found with those which result from disease in later life.

When both ears are affected, congenital deaf-mutism results. When only one is involved, we have unilateral congenital nerve-deafness. As a rule it is not recognised during injury, and it is not till childhood, or even young adult life, and often only by accident, that it is discovered. Its occurrence is often a source of considerable difficulty to the diagnostician, who is apt not to have this condition before his mind, while even if he has, it is difficult to elicit the fact that the patient "has never heard with that ear." The subject of Deaf-Mutism is considered under "Dumbness."

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Aural Diseases.—General index of articles on the ear and affections of hearing—

- **Auditory Nerve and Labyrinth.**
- **Deaf-mutism.**
- **Ear, Methods of Examination.**
  - **External Ear.**
  - **Tympanic Membrane.**
  - **Acute Inflammation of the Middle Ear.**
- **Ear, Chronic Suppuration with Complications.**
  - **Chronic Non-suppurative Inflammation.**
- **Hæmatoma Auris.**
- **Hearing, Artificial Aids to.**
- **Otorrhœa.**
- **Vertigo.**

Auto-Intoxication. *See* DIGESTION, INTESTINE, URÆMIA.

The Axilla.

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THE paramount surgical importance of the axilla lies in the fact that it contains all the nervous and vascular structures on which the power and vitality of the upper extremity depend; and consequently its diseases and injuries may lead to results apparently disproportionate to the causes to which they are attributable. For this reason the necessity of prompt recognition and early treatment is always imperative.

I. ANATOMY OF THE AXILLA

The axilla is a somewhat pyramidal-shaped space, lying between the upper and lateral part of the thorax and the upper extremity of the humerus, with its apex pointing upwards and corresponding to a spot slightly internal to the coracoid process. Its *anterior* wall is formed by the greater and lesser pectoral muscles and the costo-coracoid membrane which stretches from the latter muscle to the clavicle. The *posterior* wall is formed by the subscapularis muscle, the narrow tendon of the latissimus dorsi, and part of the teres major. The *outer* wall consists of the humerus and the conjoined origin of the coraco-brachialis and biceps muscles: while the *inner* embraces the five upper ribs, the intercostal muscles, and the corresponding digitations of the serratus magnus. Through a triangular opening at the apex, bounded by the upper edge of the scapula, the clavicle, and the first rib, pass downwards from the neck to the arm the cords of the brachial plexus and the axillary vessels. The latter are enclosed in a layer of fascia—the axillary sheath—which, following the course of the subclavian vessels, is continuous above with the prevertebral fascia and the posterior layer of the carotid sheath. The *floor* is formed by the extremely dense axillary fascia, which is continuous in front with the deep fascia covering the greater pectoral, internally with the fascia overlying the serratus magnus, and behind with the sheaths of the teres major and latissimus dorsi muscles. This floor of the axilla is held up by a fascial band which joins the axillary fascia near the border of the greater pectoral muscle, and which, extending between the brachial aponeurosis and the lesser pectoral which it invests, is continuous above with the costo-coracoid membrane. This, sometimes known as the *suspensory ligament* of the axilla, is of the greatest anatomical and surgical importance.

The contents of the axilla, which lie embedded in its loose areolar and fatty tissue, and mainly related to its walls, are the axillary artery and vein and their branches; the cords of the brachial plexus which surround the artery in the lower two-thirds of its course; and the lymphatic trunks and their associated lymph-glands. The main vessels, with the nerve cords, run in the outer wall; and this is accordingly the place of greatest surgical danger, and the point to be as far as possible avoided in all operative procedures. Important relations also exist between the branches of the axillary artery and the boundaries of the space. The thoracic axis and long thoracic are related to the anterior wall; the posterior circumflex and subscapular to the posterior wall; while the superior thoracic and anterior circumflex are in relation to the inner and outer walls respectively. Of highest surgical import is the distribution of the *lymphatic glands*. These form three groups—(*a*) the *axillary*, which, lying close to the vessels in the outer wall, receive the ascending lymphatics of the limb; (*b*) the *pectoral*, which run along the lower border of the lesser pectoral muscle close to the long thoracic artery, and receive the principal lymphatics of the mamma and the front of the chest; and (*c*) the *subscapular*, which, placed along the lower border of the subscapularis muscle, receive the lymphatics of the back. It must be remembered also, that some of the mammary lymphatics pass directly to the glands above the pectoralis minor in the apex of the axilla and under the clavicle, without joining the pectoral group (Stiles).

II. Diseases of the Axilla

Abnormalities of glandular secretion may be found, such as Chromidrosis and Bromidrosis (*q.v.*)

Superficial Inflammations, ending in suppuration, not infrequently occur. They present the same features, and must be treated on the same principles as have been laid down elsewhere (*vide* "Boils"). A special form of inflammation affecting the sweat glands has been observed here, as also about the scrotum and anal margin, situations where glands are numerous and habitually active. Small abscesses, which can be felt as deep-lying nodules, form around the sweat-coil, from the centre of which, and corresponding to the duct, a hard fibrous cord runs to the surface (*tuberiform abscess*, Velpeau). After healing, a funnel-shaped scar results, due to the cicatricial contraction of the new-formed tissue about the duct.

True or *deep axillary abscess*, lying beneath the deep fascia, may be either acute or chronic. The *acute* form is almost invariably due to glandular infection from some surface lesion, and may affect any of the groups of glands according as the point of septic absorption is situated in the hand, arm, breast, thorax, or back. Staphylococci (*S. aureus, albus, citreus*) are found in large numbers in the pus, and the disease is associated with the rapid burrowing and breaking down of tissue, common to acute septic processes. It is in this variety of abscess that the arrangement of the axillary fascia plays so important a part. The suspensory ligament practically divides the axilla into two compartments—one *pectoral* and one *humeral*. When suppuration occurs in the pectoral fossa, its extension backwards is limited by this layer of fascia, and so the abscess lies beneath the pectoral muscles, and tends to point either at the anterior margin of the axilla, or in the triangular cellular interspace between the clavicular origins of the pectoralis major and deltoid. But when the humeral fossa is invaded the pus lies in intimate relation with the axillary sheath, and tends to pass upwards into the neck along this fascial plane, causing wide infection of tissues, and even finding its way, through the upper opening of the thorax, into the mediastina. When the subscapular glands are involved the pus may burrow beneath the latissimus dorsi into the subscapular fossa, whence it may travel long distances under the influence of gravitation.

Chronic abscess may be due to lymphadenitis, to necrosis of a rib, or of the scapula, or to the extension of disease from the shoulder-joint. In the majority of these cases it will be found to be tuberculous in origin, and to present the features common to these abscesses when occurring elsewhere.

The *treatment* of acute abscess is of urgent importance. Prompt recognition of the condition and immediate operative intervention alone promise favourable results, for here, as in the neck, no abscess can be opened too early. Free incision is the only sound and satisfactory line of treatment. The arm being fully abducted, a long and deep incision should be made, the point of the knife being directed towards the inner wall of the axilla, and its back towards the outer wall, where lie the principal vessels and nerves. When the deep fascia has been freely divided, the forefinger must be thrust in and the abscess cavity thoroughly explored, and all intersecting bands broken down. In this way it is impossible to overlook, as has been done by Hilton's method, a second abscess situated in the humeral fossa. The sac should now be freely irrigated and its walls gently scraped with a flushing scoop until no more disintegrated tissue can be removed. Thorough disinfection with iodoform emulsion follows, and an attempt should be made to obtain primary union by immediate suture.

Acute Cellulitis occurs as the result of direct inoculation of a wound with the streptococcus pyogenes, or, as more frequently happens, by continuous extension from the arm. It presents the features common to all such acute inflammations: severe constitutional disturbance, redness and œdema of overlying skin, rapid infiltration and brawny induration of the parts, high fever and its accompanying symptoms. Immediate free incision must be made to relieve tension, and limit sloughing and burrowing along the fascial planes; while continuous irrigation with a warm antiseptic solution is the most efficacious method of local treatment. Strength must be maintained by nourishment, tonics, and stimulants, especially in debilitated subjects. Unless sloughing is limited by early incision, extensive and deep adhesions are likely to form during recovery, and to interfere materially with the subsequent mobility of the parts.

Tumours.—Those occurring in the axilla may be grouped into three classes—(*a*) those belonging to the infective granulomata which include syphilis and tubercle; (*b*) those originating primarily in the region; and (*c*) secondary extensions of malignant growths.

(*a*) When primary syphilitic infection occurs on the fingers or hands, the characteristic indurations are met with in the axilla, viz. multiple painless, extremely hard, slightly enlarged, easily separable, and freely movable glands. In tuberculous adenitis the enlargement is more marked, and the irregularly lobulated and caseating glands soon become matted together into a dense, indistinguishable mass, deeply fixed and adherent to the overlying tissues.

(*b*) Accessory mammæ have repeatedly been found in the axilla, either single or symmetrical, and in many cases functionally active. Lipoma is common, as also lymphadenoma and lymphosarcoma, originating in the lymphatic glands. Primary carcinoma is rare, though one case has come under the writer's notice. Notwithstanding the numerous sebaceous glands in the part, sebaceous cysts are almost unknown. This is probably due to the free movement of the arm aiding mechanically the expulsion of the secretion, and thus preventing blocking of the ducts.

(*c*) The most important tumours met with surgically are undoubtedly the secondary malignant growths. These are most frequently cancerous, following the lymph-paths from the breast; but soft, rapidly-growing, round-celled sarcomata may also cause early glandular infection, a fact which even yet is not sufficiently recognised.

In all operations for the removal of tumours from the axilla, it should be remembered that the point of greatest danger is the axillary vein, and that no danger is comparable to that of working in the dark. The first step in all cases should therefore be the thorough opening up of the space by free incision, and the second, complete exposure of the axillary vein. A useful guide to the vein is a small unnamed tributary, which, running upwards external to the lower border of the lesser pectoral muscle, enters the axillary vein at a right angle. In only one case has the writer found it entering the subscapular vein. If this vein is traced upwards and ligated close to the main trunk, the latter can be easily avoided, and all infected glandular or fatty tissue safely removed. Commencing at the highest point and working downwards, several inches of the axillary vein can be fully exposed, and by adopting this procedure there is very little danger of damaging the vein, even in the most difficult dissections. Should a wound of the vein occur, the proper treatment is temporary closure above by pressure, and suture of the vessel wall with fine sterilised catgut; as, owing to the low pressure in the vein, there is practically no tension on the stitches, and primary union quickly follows. It cannot be too strongly emphasised that in all these cases every particle of fat and fascia capable of infection must be removed, as the chances of non-recurrence are in direct ratio to the thoroughness with which the axillary contents have been cleaned out.

III. INJURIES OF THE AXILLA

The vein is the most superficial of the axillary contents, and accordingly most liable to injury from wounds. Above, it is so firmly attached to the fascia, binding it to the coracoid process and pectoralis minor, that, if wounded, it is prone to remain patent and allow the entrance of air into the circulation (*vide* "Veins"). As the result of puncture or rupture of the vessels, hæmatoma may occur, forming a diffused fluctuating swelling, rapidly followed by discoloration of the superficial parts. Rupture is most likely to follow forcible and ill-advised efforts to reduce dislocations of the humerus, and in one recorded case the subscapular artery was torn from the main trunk. In all these cases the artery, or some of its branches, is more likely to suffer than the vein, and such injuries have been followed by traumatic aneurysm. Arterio-venous aneurysm has followed wounds, involving the artery and vein simultaneously; and emphysema has also been observed as a consequence of punctured wounds. The nerves of the brachial plexus are rarely torn by traction on the limb. Such a severe injury is more likely to lead to separation of the roots from the spinal cord, as in a case recorded by Flaubert. The nerve trunks may be injured or compressed in dislocations or fracture of the neck of the humerus. The circumflex nerve from its position most frequently suffers in this way, and the associated paralysis of the deltoid is typical of the lesion. Long-continued crutch pressure may lead to paralysis, the musculo-spiral nerve and the muscles supplied by its posterior interosseous branch being specially affected. Division of a nerve trunk may occur as the result of a penetrating wound, the median being most exposed to injury. Should this occur, the wound must be at once enlarged, and the separated ends brought accurately together and sutured with every aseptic precaution. On the success of this the preservation of motor power and sensation entirely depends.

LITERATURE.—Articles "Suppuration," "Aneurysm," and "Brachial Plexus."

Azoturia. *See* Urine.

Bacteriology. *See* Micro-organisms.

Balanitis. *See* Penis.

Balneology

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Definition. — Balneology is the science dealing with the nature and uses of the various forms both of baths and of mineral springs, the name thus including more than its derivation (from *balneum*, a bath) appears to imply. Balneo-therapeutics embrace the divers manners of treatment by means of mineral waters and baths at the various spas.

In practice the use of baths and waters cannot be dissociated from the other curative influences of the Health Resort, and must be considered as necessarily connected with them. Among the subsidiary factors of spa treatment are included climate, exercise, change both in diet and manner of life, and a variable psychical or personal factor. These all may modify or participate in the proper effects of baths. Even the special procedures and skill of the bath attendants, in regard to which different spas have their own traditions, are not without importance. These local circumstances may therefore well be considered in the choice of a resort, for as much may depend upon them as on the particular bath employed. When all contributory and secondary factors of the environment are well adapted to the case in hand balneo-therapeutics find their most favourable opportunity.

The actions and uses of mineral waters taken internally are elsewhere described (*vide* "Mineral Waters"). Their effects and those of external applications often powerfully reinforce one another, and hence waters and baths are commonly employed simultaneously at many of the spas. Another balneary procedure that properly falls within the above definition is known as *Hydropathy*. The external use of water has under this name undergone, during the present century, a complete specialisation, and hydropathic treatment is now in practice, in this country, quite distinct from balneo-therapeutics. The present article is concerned with the action and uses of baths in chronic disease, as met with at the health resorts. The use of certain baths in acute disease belongs to another department of medicine.

I. Historical

The use of heat in baths dates from a very early time, and probably arose with the Aryan race in the warm climates of Asia. Similar procedures were no doubt independently employed by widely separated peoples. The history of the more common forms of bath, whether used as a luxury or for medical purposes, is not without interest. It illustrates the connection of special forms of bath with particular climates, and their influence not only on the healthy subject, but even on national character and destiny.

Hot Air Bath.—The Greeks appear to have been the first to employ baths of dry heated air. The hot chamber of the Lacedæmonians was the primitive pattern on which the later baths were founded. A sweating process was followed by frictions and aspersions of warm water. Thermal baths of water or watery vapour or heated air came to be much used as luxuries by the Greeks and Asiatics. Xenophon, describing the effeminate manners of the Persians, calls them *Balneatores*. The Romans of the Republic used only cold river and swimming baths, but introduced warm baths after they became acquainted with the Greeks. The patricians first adopted the hot-air chamber, called after its birthplace, *Laconicum*, and introduced it with various appurtenances into their own houses. Later on the State provided similar baths for the citizens at a nominal charge, and imposed upon the Ediles their oversight, and especially the regulation of temperature. These again were followed by the *Thermæ* of imperial Rome. Their remains, still extant, show them to have been the most extensive and splendid public baths on record. In the *Therma* given to the people by the Emperor Caracalla two thousand persons might bathe at once. It was characteristic of the Romans that they combined physical training and exercise with the bath, and provided for both in the same building. The bather first entered the *frigidarium* or *apopterygium*, a chamber for undressing and where frictions were sometimes applied to the skin. Thence he went on to the *tepidarium*, where he stayed until the pores were gently opened: next to one or more hot chambers,—*calidarium*,—*laconicum*, heated by a furnace, *hypocaustum*, beneath the hollow floor. Here again the bather was rubbed and sometimes anointed with oils. He now had warm water poured over him, and the skin was sponged and scraped with *strigils*. Thence again he returned to the sweating rooms, the tepidarium and the frigidarium, where he received an affusion of cold water, and was finally dried and anointed with perfumed oils.

The Roman baths extended everywhere with their conquests, and were often for convenience erected, as at Bath, in England, in the vicinity of hot springs. In Rome itself they gradually fell into disuse in the fifth century, partly from the cutting of the aqueducts by the Huns, but they continued to flourish in Alexandria, and were adopted by the Mohammedans. The Arabs brought the bath into Spain, and the crusaders still further spread its use, particularly for the treatment of the plague and leprosy. In the thirteenth century hot-air or vapour baths were common in England and in the large cities of Europe. Later on they were forgotten, but reappeared again and again, although the Celtic races retained them with some tenacity. They were brought to England about two centuries since under the eastern name *Hummun*, and once more in their present form as the so-called *Turkish Bath* about the year 1860.

Vapour Bath.—Closely allied to the baths just described, but of a much ruder construction, are the heated steam or steam and air baths employed by many different nations since early times. Such were used by the ancient Mexicans and North American Indians. The Romans utilised the steam of hot springs, and set up natural vapour baths beside the Thermæ, such as those in Ischia and the Baths of Nero at Pozzuoli. Among the northern nations the Celts of Scotland and Ireland used vapour baths, and those of the Fins and Russians remain in their primitive simplicity to the present time. A similar bath has been long used by the Japanese. In the Russian bath the heat is supplied by red-hot stones and shot. The bathers, after undressing, recline or lie upon seats that are placed around in tiers. When water is poured over the heated metal dense clouds of steam fill the room and quickly induce a perspiration. The attendants proceed to switch the body with birch twigs and rub it with bass and soap-suds. The Finlanders sometimes make use of dry heat in their baths at a higher temperature. Otherwise the Finnish bath is very similar to the Russian. Both Fins and Russians when heated and perspiring from the bath expose themselves to intense cold with impunity. The observed effects and rationale of these procedures will be discussed hereafter.

Douche Bath (Italian *Doccia*).—The Douche bath was apparently unknown to the ancients, the *stillicidium*, aspersion and pouring on of water after the Greek and Roman baths, being essentially different. The douche proper is said to have been first employed by Pietro Tussignano in 1336. Priessnitz, the father of hydropathy, utilised a mountain stream discharging from a gutter six feet above the patient's body. By the French this invaluable method of treatment has been much developed, and refinements of pressure and temperature introduced; and at the present time the *douche mobile* is at the head of balneo-therapeutic procedures in France.

Peat and Mud Baths.—It is recorded that the Romans used mineral mud baths, probably in connection with thermal springs. The peat or moorbath (German *Moorbad*) consists of an earth or mould formed from disintegrated sphagnum moss and other vegetable matter, with or without mineral admixtures. It is much employed at the Bohemian spas, and was introduced at Franzensbad early in the present century, and shortly afterwards in Germany at Pyrmont. Mud baths of different sorts have been long used in many localities, especially in the neighbourhood of warm springs and by the sea-shore. They are met with at the French and Italian spas, in the Crimea and the Baltic provinces, in Norway, in Japan and elsewhere.

The Medicinal Use of Baths is frequently alluded to in the Greek mythology. Homer speaks of Andromache preparing the bath of Hector, and of Penelope using unguents and baths to mitigate the melancholy caused by her husband's absence; and Minerva imparts strength to the weary Hercules at the springs of Thermopylæ. The beneficial effect of warm bathing in senile debility appears to have been not unknown in the same remote time. When Ulysses, after his return to Ithaca, found his father Laertes reduced to great weakness he advised him to use warm bathing, and told him that he had seen one worn down and emaciated by age, who by the use of warm baths very quickly recovered his appetite and rest. He adds that its efficacy in such cases was well known, and that it was a common custom among old men (*Odyss.* xxiv.) Hippocrates recommends warm baths in fevers, and mentions the use of hot springs, and of watery and balsamic vapours. Plato speaks of baths as useful in various diseases, as do Asclepiades and Cælius Aurelianus. The incomparable Aretæus prescribed warm sulphureous baths in elephantoid disease, and for the cure of melancholy. In using vapour baths, he first excluded the head, so as to apply heat to the body only. Celsus recommended cold baths and affusions in fevers and phrenitis, in which he was followed by Galen, who gives an excellent account of this treatment as applied to hyperpyrexia. Among subsequent writers, Oribasius, Aëtius, and Paulus Ægineta, and later on the Arabian physicians, developed the balneo-therapeutic doctrine, and extended its application. Some confined their advocacy to pure water, which they applied externally in acute and chronic diseases.

Thus Van der Heyde, treating a malignant dysentery in Holland in the seventeenth century, foreshadowed the treatment which two hundred years later was systematised as Hydropathy. Sir John Floyer, in England (1697), prominently called attention to the medical value of baths, but their use is scarcely mentioned by Sydenham, and it is discouraged by Mead. The opprobrium into which baths had fallen in this country partly arose "from the notion of the moderns that *macerating* in hot water and *sweating* in bed afterwards answered all the purposes of bathing" (T. Glass, 1752). Both waters and baths were, however, prescribed by Boerhaave and by Heberden. Sir James Currie, at the end of the eighteenth century, in his *Report on the Effects of Water, Cold and Warm,* may be said to have laid the foundation of exact balneology. His work at once awoke a strong interest in the subject in Germany. The doctrine of hydrotherapy was revived. The empiricist Priessnitz began his water treatment in 1829. Under a fresh impulse of medical inquiry the mineral waters were everywhere analysed and taken into use; the douche bath was introduced into France, and Peat baths into Austria. The latter half of the nineteenth century has witnessed the application of exact methods and systematic study, and the commencement of a new fabric of balneology. But the Vienna school of scientific hydrotherapy led by Winternitz, by establishing the rationale of common balneary procedures, marks the most important advance of the century.

II. The Action of Baths

Differences not only in the *ingredients*, but in the *vehicle* and *physical properties* of baths, affect their action. Besides liquid baths some, like peat and mud baths, are semi-solid; some consist wholly, or in part, of vapour, or of gases of various kinds, such as carbonic acid gas or atmospheric air; and others of solid particles, as the sand bath. In all these cases, particles of matter in the liquid, gaseous, or solid form are the vehicles for conveying energy to the body or abstracting energy from it. Energy is also applied directly without material intervention, in the same manner as the body discharges heat, by radiation. In the "sun bath" and artificial imitations of it the skin is exposed to the action of the solar or other rays. Baths of all kinds are applied either to the entire surface of the body or locally. One form may be combined or alternated with others, and differences in the precise form of the bath, its temperature, duration, and attendant circumstances, greatly modify the resulting action.

In whatever form applied, baths exert their action solely upon the skin. That is to say, they take effect *directly* upon the skin, and through the skin *indirectly* upon other parts of the body. The chief value of a mineral bath was formerly supposed to attach to the cutaneous absorption of its ingredients. Saline, alkaline, or muriated waters were believed to produce essentially the same effects, whether internally or externally applied. This opinion, based on a mistaken view of the functions of the skin, is now known to be erroneous. It has been shown that mineral matters dissolved in baths do not pass *through* an unbroken skin, and that even pure water is only absorbed in small quantities, and that absorption is a negligible factor in most cases. Certain gases and volatile matters, such as sulphuretted hydrogen and aromatics, occur in baths, and may pass through the epidermis. If frictions are used in connection with baths the cutaneous absorption is powerfully aided, and volatilised or dissolved matters may pass into the circulation, as in mercurial inunction. Apart from these exceptional cases, the chief action of baths is the application at the body surface of heat and moisture, and of different forms of mechanical and chemical energy.

Hence it is that the physiology of the skin is at the foundation of balneology. A knowledge of the intimate connections subsisting between the skin and other parts of the body is necessary, in order to a correct appreciation of the effects of baths in chronic disease. The chief relative physiological propositions may be summarised as follows:—*Firstly*, the skin as a heat-regulating organ: The low conductivity for heat of the skin and subcutaneous fat prevents too rapid a loss of bodily warmth, and permits life to go on in temperatures far lower than that of the body. Of the daily loss of heat, estimated at 2,500,000 calories for a healthy man, about nine-tenths passes by radiation, conduction, or evaporation from the skin (Vierordt). The loss of heat at any time is proportional to the excess of the skin temperature over that of the external medium. The warmer the skin, the greater is the loss of heat, and, on the contrary, cooling of the skin, by contraction of its blood vessels, diminishes the loss and conserves the bodily warmth. The blood supply of the skin is of such a kind, and governed by a mechanism so sensitive, that the loss of bodily heat is adjusted moment by moment to the fluctuations of external temperature. *Secondly*, as a depurative organ: The skin is furnished with a vast secretory and excretory glandular system. *Thirdly*, it is by the skin that relation is had with the external world. An expansion of the nervous system is spread over its surface, by means of which different forms of sensation are habitually conducted to the brain. There is also reason to believe that certain areas of the skin are related to corresponding internal organs, and that both are consentaneously affected, and one by the other (Head). Such a relation between deep and superficial parts goes far to explain the influence of sedative or stimulating cutaneous applications in visceral disorders. A general connection between the skin (external superficies) and the mucous membranes (internal superficies) is probable on developmental grounds, and is confirmed by experience of the effects of baths in gastro-intestinal catarrh. Evidence is also furnished by the distribution of exanthems, and the internal lesions—intestinal, pulmonic, nephritic, etc.—met with in superficial burns. The existence of such a connection was affirmed by Hippocrates in the aphorism: *alvi laxitas cutis densitas, cutis raritas alvi densitas.*

Of the various elements—heat, moisture, chemical and mechanical stimulation—that enter into the action of baths, heat and moisture are common factors, the effects of which upon the several functions of the skin can be definitely stated. Chemical and mechanical stimulation belong to certain forms of baths, and will be considered in connection with them.

EFFECTS OF HEAT—(*a*) *On the heat-regulating function.*—The effect produced on the bodily heat varies with the temperature of the bath. There is no change in heat production or loss when the body is immersed in water at 93° to 95° F. This is therefore stated to be the point of thermal indifference for aqueous baths. Baths at about 93° F., of from 15 to 25 minutes duration, may therefore be called *athermal* in their effect on the body. At lower temperatures baths increase the loss of heat in proportion to their coolness, and sometimes to a very great extent. The production of heat in the body is, however, correspondingly augmented, and in a cold bath it may be increased three or four-fold (Liebermeister). Cold applications do not, therefore, lower the bodily temperature in normal conditions of heat production, unless the bath be much prolonged. Baths below 93° F. are *hypothermal* in their effect upon the body. They differ from baths at a higher temperature in the same way as minus differs from plus, and have an important therapeutic range. Baths at a temperature above 93° may be conveniently designated as *subthermal* from 93° to 98° F., and *thermal* from 98° F. and upwards. Such baths produce an accumulation of heat by checking its normal loss. When the loss of heat is entirely prevented the body temperature may rise as much as 4·5° in half an hour (Liebermeister, quoted by Leichtenstern). Hot immersion, and vapour baths in particular, rapidly augment the body heat and produce an artificial pyrexia.

(*b*) *On tissue change.*—Applications of cold, which increase the loss and production of heat and the excretion of carbonic acid gas, are believed to augment the conversion of fat (Voit). Experiments have shown that thermal baths, on the other hand, which check the loss of heat and raise the body temperature, increase the metabolism not only of the fats but of the nitrogenous tissues, with increased excretion of urea (Leichtenstern, Godlewsky). Such a result may indeed be expected on *à priori* grounds to arise in a condition of artificial fever. A full elucidation of the effects of the various kinds of baths on tissue change, and especially of the local application of cold and heat, is still desiderated. Baths increase the glandular activity of the skin in proportion to their temperature. In a hot bath the quantity of perspiration is greatly increased, with a proportionate loss of body-weight.

(c) *On blood-vessels and circulation.*—It is not surprising that baths should exercise a powerful effect on the circulation and distribution of the blood if it be remembered that they operate on the most extensive vascular area of the body. The temperature of the skin in any part depends on the amount of arterial blood circulating in it. In different parts of the body the superficial temperature in health is commonly from 5° to 12° F. or more below that of the internal organs (Kunkel). Hence it is that, as sensations of temperature are derived wholly from the skin, baths a few degrees below blood-heat generally feel warm.

1. In baths at a temperature lower than that of the skin a protective contraction of the peripheral vessels takes place ("increased vaso-motor tone") proportionate to the difference of temperature. There is at the same time a rise of blood-pressure and increased force of the cardiac systole. The pulse is at first accelerated and then retarded (Winternitz). Increased tonus and diminished blood supply in the superficial parts, or those exposed to cold, are accompanied by a corresponding diminution of tone and increase of blood supply in the viscera, or in parts not so exposed. The circulation is therefore to some extent short-circuited. In the rabbit dilatation of the vessels of the *pia mater* has been observed on application of cold to the abdomen (Schüler). Similarly a cold sitz bath increases the circulation in the upper parts of the body (Winternitz). The vascular tonus quickly reaches its maximum, and ultimately relaxes if the exposure to cold continue, with a rapid loss of internal heat. The relaxation of the capillaries, whilst the arterioles and venules are still contracted, is indicated by a blue-red colour of the skin.

2. In warmer baths, at a temperature at or above that of the skin, but below blood heat, the action on the circulation is entirely different, and baths of this kind, especially when combined with various forms of cutaneous stimulation, have a high value in cardio-vascular disorders. Their general effect is tranquillising and sedative, they withdraw heat to a moderate extent, and, by diminishing the vaso-motor action of peripheral parts, they invite the blood into the extremities and the skin.

3. Lastly, the effect of thermal baths is to open the surface arterial system, to accelerate the pulse, and diminish the general blood-pressure. This condition may continue for many hours after the bath, with a degree of enervation and debility proportional to its temperature and duration. It is remarkable how tolerant the body is of sudden and extreme changes of temperature when the cutaneous circulation is opened by recent thermal stimulation. The Russian comes out of his sweating-chamber at 130° F. with a red-hot skin, and rolls in the snow or in iced water with impunity. These cold applications after heat restore tone, raise the blood-pressure, and prevent the enervation and debility just referred to.

The reaction of the blood-vessels both to heat and cold has been generally regarded as reflex, through the vaso-motor centre. Whilst this is no doubt true in the case of sudden and intense stimuli, such as that experienced on entering a cold bath, there is reason to believe that a local and direct reaction on the part of the contractile peripheral arterioles (the so-called *cœur periphérique*) also takes place. It is held that stimuli of cold and heat, especially those of a gentle and continuous character, produce locally in the vessel walls the movement of dilatation or contraction. Not only so, but the activity of the unstriped muscular fibres, whether of the blood-vessels, intestine, or bladder, is directly responsive to the application of heat or cold (Onimus).

(*d*) *On the nervous system.*—The influence of different degrees of heat on the nervous mechanism governing glandular activity and vascular tone has been already alluded to. It remains to consider in what manner the temperature of baths affects the skin as an organ of sensation, and to what extent the nervous centres and the body at large are affected mediately through the skin. The *sensation* of bodily warmth depends on the fluctuations of cutaneous arterial supply, is relative in character, and in baths closely follows changes of temperature. (1) The impression of cold in cool or cold baths stimulates the respiratory and vaso-motor centres, and excites reflex muscular contractions and increased production of heat. (2) Baths slightly below blood-heat, and somewhat warmer than the skin (subthermal), produce a diffused sensation of warmth, in the same manner as frictions. On the whole, peripheral excitation is diminished and equalised. This is for most persons the pleasurable degree of heat. It is this mild, continuous, thermal stimulation of the skin which is alluded to in the psychological definition—"Heat is a typical form of passive physical pleasure" (Bain). The massive sense of warmth, the titillation of so large an area, and the undulatory movements of the water, have a calming and soporific effect on mind and body. Subthermal baths are therefore usually sedative. (3) On the other hand, baths above blood-heat, like those considerably below it in temperature, have an initial stimulant effect on the nervous centres. If the application of heat be continued, the muscles, both voluntary and involuntary, are relaxed, and the circulatory excitement rapidly becomes febrile, with profound enervation and ultimate paralysis. These secondary and febrile effects of heat are not produced, even by the hottest thermal baths, if these are sufficiently brief, or followed by an adequate application of cold.

It cannot be doubted that stimulant or sedative actions produced on the skin are participated in, through the nervous centres, by all parts of the body. Whether the nervous end-organs, and therefore the sensibility of the skin, be moderately and pleasantly affected, or more powerfully stimulated, by cold or heat, corresponding activities are produced elsewhere which affect the course of trophic and other tissue changes as well as the various secretory and excretory functions. The exact nature and sequence of these indirect activities have yet to be defined and elucidated; but the doctrine of the general action of baths through the peripheral sensory nerve-organs is the corner-stone of balneo-therapeutics.

Effects of Moisture.—In most baths the action of heat and of moisture on the skin are conjoined. The first may be considered in general a stimulant, and the second a sedative influence, and the general character of the bath depends on the predominance of one or the other. Moisture softens and relaxes the skin, separating the dead epidermic scales and causing the deeper cells to swell by imbibition of water. Massage is often more effectual in connection with baths because it operates upon mollified tissues.

Effects of Pressure.—In all immersion baths the body is subjected to a pressure additional to that of the atmosphere, and proportional to the depth and specific gravity of the bath. This pressure, unless counteracted by other agencies, causes contraction of the peripheral vessels and tends to drive the fluids to the deeper parts. It also hinders the respiratory movements. It has been estimated that the total additional pressure on an adult body in a bath of water amounts to from 500 to 600 kilogrammes. These facts have a bearing on the use of baths in circulatory disorders.

III. The different Kinds of Baths and their Actions.

1. Indifferent Thermal and Subthermal Baths.—Bath may be taken as typical of thermal, and Schlangenbad of subthermal, spas. The therapeutic effects of this large class of baths are mainly those of heat and moisture, which have been already discussed. No doubt the physical, and possibly the electrical, properties of the water and the presence of alkaline or siliceous, and especially of gaseous constituents, may modify the action. Thermal baths are commonly described as varying in temperature from 80° to 105° F., but the division here made into *thermal* and *subthermal* (viz. above or below blood-heat) corresponds with an important practical distinction. Many so-called "thermal" baths, like those of Schlangenbad and Nauheim are not really thermal in their effects, but antiphlogistic or cooling. At most of the European spas the duration of thermal baths, which are seldom above 105° F., varies from ten to thirty minutes. Perhaps the commonest error in spa practice is to misapply baths above blood-heat. The debility that sometimes follows a course of such baths in delicate persons may not be easily overcome. This has given rise to the erroneous opinion that immersion baths cannot be safely taken by invalids many degrees above 100° F. In truth, far more depends on *duration* than on temperature.

In the hottest thermal baths, such as those of New Zealand and Japan the bather enters the water at 130° or 140° F., but immersion can be tolerated for such brief periods (in Kusatsu, three minutes only) that the later and enervating effects of heat are not developed. On the other hand, subthermal baths may be continued for two, three, or even six hours, and daily repeated, as at Leuk in Switzerland, with great advantage in appropriate cases, and without subsequent debility. At Kawanaka, in Japan, the bathers remain in the water, which is a little below blood-heat, for a month on end, with stones on their laps to prevent them from floating in sleep; and it is said that the caretaker, a hale old man of seventy, stays in the bath the entire winter.

What has been described by the French as *Fièvre thermale*, or "critical fever," is frequently met with under treatment by indifferent warm baths. After a certain number of baths have been taken, a certain languor or malaise is experienced, with muscular stiffness, and recrudescence of former symptoms, and occasionally some form of cutaneous eruption (the so-called *poussée*). These phenomena, to which great value was formerly attached as a critical and favourable event, are now regarded as indicating an excess of thermal treatment and necessitating its intermission.

(*a*) *Thermal* indifferent baths have a limited, but valuable, application in balneo-therapeutics. They produce a stimulation proportional to the degree of heat, but, unless of brief duration, followed by corresponding debility. The spa physician may well bear in mind the saying of Hippocrates: "A bath enfeebles every time that its heat exceeds that of the body immersed in it." Brief thermal or even hyperthermal baths may be used to stimulate the cold and dry skin, or inveterate eruptions; for inertia or torpor, either of the nervous centres or of the circulation, for diminished sensation or feebleness of muscular movement, and for some chronic painful disorders of the muscles, nerves and joints.

(*b*) *Subthermal* baths have a very large place in spa treatment. They usually exert both sedative and stimulating influence. The heart's action is steadied and slowed (see previous section), sometimes as much as twenty beats per minute; peripheral nervous excitation is diminished, but a massive and equable tonic impression is produced. These baths are therefore valuable in conditions of irritability of the vascular and nervous systems, as well as in debility and motor weakness, in peripheral and incipient and reflex paralyses. They promote absorption of inflammatory exudations and congestions (in the peritoneum, joints, etc.); they allay disturbances of nutrition, moderate senile change (*senectus ipsa morbus*), and are much used in degenerative arthritis, in atonic gout and rheumatism, and in conditions of nervous exhaustion and of convalescence.

2. DOUCHE BATHS.—All that has been stated with reference to the effects of heat, and to degrees of thermality, applies with equal force to the douche. Thermal, subthermal, and hypothermal douches have within certain limits the same therapeutic application as the corresponding immersion baths. The chief differences between the two forms of treatment depend upon the posture of the body, upon hydrostatic pressure, which is a considerable factor in the immersion bath but absent from the douche, and upon the presence in the latter of the mechanical stimulation of impact.

Moreover, the douche furnishes various modes of purely local treatment.

(*a*) The hypothermal (cool or cold) douche is employed either as a general tonic, or locally as a sedative in inflammatory or febrile conditions, and has been used by the French for chronic neuralgia.

(*b*) The thermal douche is only applicable to general disorders or local conditions of an asthenic character. It is a powerful stimulant, arousing dormant nutritive processes. It is therefore valuable in chronic glandular engorgements, in congestion and inaction of the abdominal viscera, and in degenerative conditions of the joints; also in nervous debility and torpor, in paralysis, with little or no structural change, and in some cases of anæmia and inaction of the skin. In rheumatism and gout it should never be prescribed until the inflammatory stages have quite subsided.

(*c*) The subthermal douche is a valued remedy for degenerative ("rheumatoid") arthritis, and for many other chronic joint affections. Considerable importance attaches to the degree of pressure used in the douche bath. A powerful impact of water is no doubt beneficial in some cases as a general tonic, especially to the muscular system. It should, however, never be employed in a routine manner. The high-pressure douche is not at all adapted to conditions of vascular or nervous debility, especially when applied to the trunk. On the other hand, large volumes of subthermal water *at little or no pressure* produce a grateful and sedative effect, and can be safely and properly applied in conditions of great weakness and in many cases of organic heart affection. Massage can be combined with all forms of the douche, as at Aix-les-Bains. It is probably more effectual in connection with baths, because the tissues are mollified and the superficial layers of epidermis are readily removed. Here, again, the vigour of the application should be carefully adapted to the requirements of the case. The douche and douche-massage are potent agencies, and the regulation of temperature, pressure, and the amount of manual stimulation cannot safely be left to the discretion of attendants.

The position of the patient is also a point of some little importance, and a reclining posture is advisable in some cases. Among special forms of the douche may be mentioned the *Douche Écossais* in which the temperature of the water constantly alternates between hot and cold, the needle, spray, shower, and wave douche, and the spinal douche, also the "submarine" douche in connection with immersion baths. The douche can be used in a great variety of ways locally, for example the so-called "ascending" douche, as an application for hæmorrhoids, prolapsus ani and uteri, and for uterine congestion, leucorrhœa and dysmenorrhœa. Hypothermal local douches are often of advantage in local inflammatory states; and for the opposite condition of feeble and degenerate nutrition, with exudation, stiffness, and swelling, the thermal douche is equally appropriate.

3. Peat and Mud Baths.—(Franzensbad, Dax). (*a*) The peat or moorbaths of the Bohemian spas contain a considerable quantity of iron (from 7 to 15 per cent of the dried material) as well as some free sulphuric and other acids. In addition to the thermal element, the contact and pressure of the semi-solid mass on the body affect the circulation and nerve endings Peat baths are believed to act in virtue of a special stimulation or excitation of the skin, both mechanical and chemical. They are prescribed in torpidity of the skin and circulation, in superficial neuralgias and neuritis, in disorders of common sensation, in chronic rheumatic affections, and to promote the absorption of morbid deposits. Their use is contra-indicated in excessive nervous irritability and serious vascular degeneration. As the specific heat of the peat bath is less than that of water, a comparatively high temperature can be borne without undue constitutional disturbance. Peripheral stimulation is decidedly more active in the peat bath than in water at the same temperature. There is, however, in peat baths a greater sense of pressure than in water baths, which makes it wise in most cases not to cover the upper part of the chest. Besides those in Bohemia (Franzensbad, Marienbad, etc.), the principal peat baths are met with in Germany (Pyrmont, Schwalbach, Elster, Driburg, etc). Similar baths are used at Lipetsz in Russia, and Strathpeffer in Scotland.

(*b*) Mineral mud baths (French *Bains de Boue*) are of very various kinds. In many of them, such as those at Sandjford in Norway and in the Crimea, the principal constituent is common salt. Others are composed of the mineral and organic deposits from thermal springs, like those at Dax and the Italian spas. Many contain iron or sulphur and arsenic in appreciable quantities. The action of these baths depends not only on their temperature and constituents, but on the special ways in which they are employed. Some are applied with frictions (Sweden), and many are used for the most part locally to diseased parts (Battaglia). The brine or marine mud baths have been especially recommended in rheumatism, in old syphilis, and in contractions, the result of wounds. Among the spas where mud baths are employed may be mentioned Dax, St. Amand, and Balaruc in France; Meinburg, Nenndorf, and Kissengen in Germany; Acqui, Battaglia, Vinadio, and others in Italy; Laurvik, Modum, Sandjford in Norway; and Loka in Sweden. They are also used in the Baltic provinces and on the Black Sea, in New Mexico (Las Vegas), in Japan, etc.

4. Vapour Baths.—(It. *Stufe*, Fr. *Étuves*).—A natural vapour bath is provided wherever warm vapours issue in a grotto or cave, or are artificially led from their source or from the surface of thermal water to an appropriate chamber or "bouillon." In temperature they vary from 80° to 120° or 130° F. Above that heat aqueous vapour is intolerable. The hot vapour condenses on the surface of the body with the development of heat, but without the pressure effects of ordinary baths. Perspiration is rapidly induced, the epidermis softened, and the circulation and nutrition of the skin increased. The pulse is increased in fulness and frequency, and, if the head is included, there is corresponding fulness of the pulmonary capillaries. It is held that these baths promote the resolution of lymphatic engorgements and remove indurations and concretions in the neighbourhood of joints. They also help to soften the harsh, hard, thickened, and inactive skin, and promote the

healing of sluggish ulcers. The influence on the nervous centres varies with the temperature. It is sedative if the effect of moisture predominates over that of heat. If the contrary be the case an unpleasant excitement and elevation of temperature occur, with subsequent malaise. It has been well said that the heat of a vapour bath (as, indeed, of any other thermal application) should bear an inverse proportion to the heat or febrile excitement in the patient. Assolini used vapour baths below blood-heat in inflammatory affections of the throat and respiratory passages. In subacute catarrhs and chronic affections a more stimulating vapour can be employed, such as those of Plombières or Mont Dore. Natural vapour may be locally applied by various apparatus (*e.g.* the "Berthollet baths"). Some of the most remarkable natural vapour baths are met with in the island of Ischia, such as that of San Lorenzo (124° F.) and Castiglione (133° F.). The *Bagni di Nerone*, near Pozzuoli, yield a vapour at 167° F., which was led by the Romans into large chambers. The neighbouring *solfatara* emits a sulphurous vapour. Lucca, Monsummano, and Battaglia in Italy, and Cransac in France, also possess natural vapour baths. Among European spas, at which the vapour of thermal waters is employed in the same manner, may be mentioned Aix-la-Chapelle, Plombières, Baden, Luchon, and Bath in England.

5. HOT AIR BATHS ("Turkish bath," *Étuve sec*, local hot air bath).—The toleration of the body for the extreme temperatures (170° to 200°, or even 240° F) of the hot-air bath depends upon the fact that the latter interferes in no way with the heat-regulating function of the skin. Whilst the vapour bath checks evaporation, and soon raises the heat of the body, the hot-air bath favours evaporation, and may be continued for an indefinite period without sensible elevation of the internal temperature. The *superficial* temperature is, however, raised and the cutaneous circulation greatly increased. The limbs are also increased in volume by the blood drawn from the deep parts, and the general blood-pressure falls. The excess of blood drawn through the peripheral vessels supplies a flow of perspiration which may amount to two pounds in weight during the course of a single bath. The centrifugal flow of fluids is favoured by the circumstance that in a hot-air bath the external pressure on the body is less than that of a normal atmosphere, which is the reverse condition to that which obtains in ordinary baths. The skin is macerated and cleansed by the removal of epidermic accumulations and gland contents. The subsequent application of massage and cold affusion cause contraction of the blood-vessels and rise of blood-pressure. In addition to their time-honoured hygienic value, hot-air baths enjoy a wide therapeutic application. From their powerful effect on the peripheral circulation they are prescribed for persons of a sluggish, dry, or cold skin, especially for those who complain of want of animal heat and who frequently "take cold." Such patients are very generally unable to take cold affusions unless the skin has been previously heated and vascularised by hot air or other non-debilitating thermal application. These baths relieve some cases of embarrassed venous circulation, especially splanchnic congestions or obstructions with irregular distribution of the blood; also chronic renal affections and gouty and rheumatic disorders, by reason of their eliminative action.

The *local application* of very hot air is often a valuable auxiliary to ordinary bath treatment. By applying suitable apparatus to the part, or including it in a hot chamber, temperatures of 200° to 300° F. or more can be readily brought to bear locally on the affected limb or region. A very powerful thermal action is thus produced with the object of modifying the circulation and the nutrition in a limited area. Such applications, limited to a single limb, commonly raise the body temperature from 1° to 3°. Experiment has shown that whilst the general body temperature of a dog was raised 1·8°, that of the venous blood returning from the limb rose 4·8° in half an hour (W. Bain). Local superheated air baths are of various kinds, and are prescribed in order to stimulate the process of repair in degenerate tissues, the absorption of swellings and exudations, and the removal of stiffness and contractions.

6. MURIATED (BRINE) AND MARINE BATHS.—It is believed that in muriated baths some salt passes through the epidermis and affects the nerve endings. The stimulant action of these baths is found to be proportional to the strength of the brine. The deposit of saline particles on and in the tissues occasionally causes eczema or urticaria in persons of a sensitive skin.

(*a*) Brine baths (Droitwich).—Nearly saturated brines like that of Droitwich may contain as much as 30 per cent of salt. They are employed as a stimulating balneary treatment in "muscular rheumatism" and sciatica, in chronic articular affections, and as a tonic in convalescence. Kreuznach in Germany, Salins in France, Ischl in Austria, and Bex and Rheinfelden in Switzerland, possess baths of this class.

(*b*) Marine baths (Margate).—Different seas vary in the proportion of salt. The Atlantic water contains about 3 per cent, besides free gases. The tonic effects of open sea- or surf-bathing depend partly on the fresh open air, marine climate, and the movement of the waves. Sea baths increase the resistance of the skin to changes of temperature and to damp, and therefore tend to prevent catarrhal and rheumatic affections. They are beneficial in many atonic conditions, whether dyspeptic or nervous, in anæmia and chlorosis, asthma, chronic catarrh, and scrofulous and rachitic affections. They are contraindicated in eczema, erysipelas, phthisis, nervous erythism, and "bilious" or hepatic dyspepsia. Sea-water may sometimes be employed with advantage in the form of douches, as at Sidmouth. *Sea-weed baths* of a semi-liquid consistence prepared from the crushed bladder-wrack (*Fucus vesiculosis*) are used at many marine resorts (Nairn in Scotland, Margate, etc.).

7. GASEOUS MURIATED BATHS (Germ. *Soolbäder*) (Nauheim).—Some muriated waters contain free carbonic acid gas in sufficient quantity to affect the skin as a mechanical stimulant. Even a subthermal or tepid bath of this kind gives quite a warm and stimulating impression. It is therefore possible, by means of effervescing baths (both of the muriated and of the chalybeate class) to combine the tonic and sedative and antipyretic effect of cool temperatures with an active peripheral stimulation. It has been already shown that ordinary subthermal baths strengthen and slow the heart's action and open the peripheral circulation. The addition of salt and free gas accelerates and enhances this peripheral effect, with increased redness of the surface, expansion of the capillaries, and a corresponding sense of warmth. Besides the direct effect thus produced on the distribution of the circulating blood, it is believed that indirect effects on the heart and other organs are produced through the vagus, vaso-motor, and other nervous centres (see under Section IV. "Heart Affections"). If the temperature of the bath is reduced below a certain point peripheral expansion is antagonised by the vaso-constrictor effect of cold, and blood-pressure rises. In addition to cardiac affections, these baths are employed for their sedative and tonic action on the nervous centres. Graduated Swedish exercises are frequently taken in connection with these baths. The latter can readily be artificially prepared and administered in the patient's own home, but not

with the same advantage that attaches to the favourable circumstances of the health resort. "Artificial Nauheim baths" usually contain two to three per cent of salt and two to three *per mille* chloride of calcium, and sufficient alkali and acid to make them effervescent. The weaker bath is first used at 95° F., and for subsequent baths the temperature is gradually diminished and the saline strength increased. Soolbäder, similar to those of Nauheim, are given at Oeynhausen and Soden in Germany, and are artificially prepared at many of the British spas.

8. SULPHUR BATHS.—Most of the numerous cold and thermal sulphur waters are employed externally in the form of baths. Some importance appears to attach to the combination in which the sulphur element occurs. Sulphuretted hydrogen is readily absorbed through the unbroken skin, but not alkaline sulphides. Again, the associated constituents of the water, as in muriated sulphur waters, powerfully affect the action of the bath. Pure sulphuretted waters, like those of Weilbach or Strathpeffer, are employed as baths in eczema and other skin affections, in subacute rheumatism, the results of rheumatic fever, and in gout. The thermal sulphur waters, like those of Luchon, are also much used in skin affections and in rheumatism, but their content of sulphur is so small as to make absorption improbable. The use of the baths, however, tends after a time to increase or revive old eruptions. At Aix-la-Chapelle they are habitually prescribed in cases of syphilis to gauge the activity of the specific infection and to promote the elimination of toxins. Some of the very hot thermal sulphur baths of Japan, which are also much used in syphilis, contain considerable quantities of free sulphuric acid, which increases their stimulant action on the skin. The same remark applies to one of the baths at Rotorua in New Zealand.

9. PINE BATHS.—Among other aromatic preparations an extract of pine leaves is often made, and employed as an agreeable addition to the warm bath at many of the spas.

10. SAND BATHS.—In the old procedure of arenation the body, or a portion of it, is immersed in sand naturally or artificially heated to from 110° to 130° F. Profuse perspiration is caused, with redness of the skin and crusting of the sand upon it. The normal loss of heat is therefore hindered, and the body-heat may rise as much as three or four degrees. These baths are recommended in cases where a profuse diaphoresis is required, as in some chronic kidney diseases, and for their local effect in joint affections, and especially in degenerative arthritis. They have been employed from ancient times in the island of Ischia, and are used at Lavey in Switzerland and elsewhere.

11. SOLAR AND RADIATION BATHS.—The practice of exposing the body to the solar light and heat dates from remote times, and the modern "sun bath," which is employed with undoubted advantage in many sanatoria, is but a revival of it. The use of electric radiation has been recently introduced and can be quite readily applied, both locally and generally, as a "radiant heat bath." It is undeniable that a powerful thermal effect is produced on the enclosed part; but in what way the differing forms of energy contained in the solar or electric radiation affect the tissues has yet to be accurately determined.

IV. GENERAL BALNEO-THERAPEUTICS

Apart from the common *hygienic* use of baths for the maintenance of health and the prevention of disease, baths have a wide *medical* application in the treatment both of acute and chronic affections. In acute disease they are most often employed for their temperature effects, for example, in pyrexial and inflammatory conditions, in cramp, convulsion, and other nervous affections, in failure of the heart, whether from syncope or venous engorgement, and in other conditions of profound debility. They are also used in acute diseases of the skin and of internal organs, simply as vehicles for the external administration of drugs. But chronic maladies—the various conditions met with in those seeking treatment at the health resorts—are the proper field of balneo-therapeutics.

Following the order adopted in a previous section, the use of baths will be considered in affections of the skin and circulatory organs, in disorders of nutrition and elimination and in affections of the nervous system.

1. Skin Affections.—Many common functional abnormalities of the skin are favourably affected by a course of baths. The inactive, torpid, and often chilly skin is stimulated by brief hot baths (from 105 F. upwards), and by alternating hot and cold douches. When the glands are obstructed and epidermis accumulated, hot-air or vapour baths are indicated. For the ill-nourished cachectic skin, and the dry, papery, atrophic skin of the old, warm soft-water baths, and especially sulphur and alkaline baths, are helpful, and the massage douche at a temperate heat. The same baths may be prescribed in pruritus. The use of sulphur waters both internally and externally is peculiarly appropriate to skin affections. Psoriasis and Ichthyosis may be relieved by hot sulphur baths, by hot air and vapour and by prolonged subthermal baths. Most forms of eczema are benefited by baths. If the eruption be recent and moist the alkaline or sulphur bath at an indifferent temperature is indicated. With localised and dry eczemas a more stimulating and hotter bath can be used. Eczema, acne, furunculosis, and other conditions of the skin dependent upon underlying disorders are equally amenable to this class of remedies, but often exhibit temporary exacerbations under their use.

2. Affections of the Cardio-Vascular System.—(*a*) The chilly skin, the common condition of contracted cutaneous vessels, usually associated with catarrh (or indigestion), deficient oxidation and want of bodily heat. The natural distribution of the blood is disturbed and the action of the heart is usually languid. Stimulating thermal treatment is here indicated, such as hot peat baths or thermal douches with massage, or hot air.

(*b*) Irregular vaso-motor conditions, such as those met with at the menopause. Temperate and subthermal baths exert an equalising and sedative influence not only on the peripheral circulation but also on the nervous centres.

(*c*) Sub-inflammatory states of the small subcutaneous veins and connective tissues common in gouty persons. Any stimulating thermal treatment, especially if combined with massage, may readily induce inflammatory stasis with patches of phlebitis and painful swellings. Eliminative treatment is usually indicated, by waters or otherwise, and only sedative baths or applications are permissible.

(*d*) Heart affections.—A clear distinction must be made between *existing* morbid conditions of the endo- or myo-cardium and signs which are merely evidences of *bygone* disease. Wherever indications are presented of existing heart affection of any kind, much care is requisite in the use of baths, both as regards temperature and duration. In structural heart disease, the heat of the bath should seldom equal that of the blood and never exceed it. In such cases, balneary treatment is essentially subthermal (*i.e.* from 80° to 98° F.), and can be effectually carried out by more than one procedure. One of the best is the *subthermal douche at low pressure*, without the excitement of impact, and accompanied by skilled superficial massage. Immersion baths at a similar temperature, whether plain or muriated, or muriated and gaseous as at Nauheim, have essentially the same effect, allowing for difference of posture. (See Section III. "Gaseous Muriated Baths.") All baths, whatever the temperature reading may be, that give the patient the sensation of heat must be avoided. An attendant should always be present throughout, and the physician should observe for himself, at all events the effects of the first bath, in every case. The employment of surface massage with the douche and the use of carbonated gaseous brines for the immersion bath, by reason of the continuous peripheral stimulation they produce, enable comparatively low temperatures, from 95° down to 85° and even 82° F. to be readily borne without sensation of cold. During the bath the pulse should become stronger and slower, and the breathing deeper. If at any time it begins to accelerate, the breathing become embarrassed or the facial colour change, the bath must be discontinued. Grave injury is done by overheating the patient in these cases. No hot packs, close dressing-rooms, or the slightest subsequent fatigue are permissible. Chronic rheumatic or gouty carditis, commencing degeneration and dilatation, with or without valvular affection, aortic or mitral; weak, frequent, and irregular action from overloaded and embarrassed right ventricle, and many phases of disordered innervation and faulty nutrition, the results of anæmia or debilitating illness, are sometimes notably benefited by a suitable course of baths if these rules are duly observed.

3. Disorders of Nutrition and Elimination.—Both waters and baths are favourable to elimination, for it is among their primary and most familiar effects to stimulate the excretory activity of the kidneys and the skin. It has been already seen that cold and subthermal baths and douches affect nutrition by increasing tissue change and oxidation. For particular failures of elimination and specific defects of nutrition in the various tissues balneo-therapeutics provide a variety of treatment, the effectual application of which taxes the knowledge and experience of the spa practitioner. The recognition of the part played by toxins in many disorders emphasises the importance of eliminative treatment. In many conditions of low health, some or all of the excretory functions are at fault. How common is chronic inaction of the bowels and of the skin! Again, persons who lead a sedentary and artificial life, it may be with habitual nervous over-expenditure, maintained by a stimulating diet, frequently find that a periodical course of eliminative treatment at one of the spas maintains them year by year in average health and prevents or retards the development of family diseases.

(*a*) *Digestive Disorders.*—In those common defects of nutrition that are traceable to catarrh of the digestive mucous membrane, and feeble secretory and muscular power of the same, waters and baths are often beneficial, and should always be combined with an appropriate diet. Many forms of mineral water, but especially the weak muriated, and muriated-alkaline, and the nearly pure or effervescent waters, may be used internally. In cases where waters are ill borne, benefit often results from thermal and subthermal baths. The massive sedation or gently stimulating action produced on the skin surface appears to be reflected on the mucous surface (*vide supra*). More stimulating applications, such as the *douche Écossais*, may be gradually added. Gastric neuroses and other forms of painful dyspepsia, with emaciation, are peculiarly amenable to this treatment. The use of sulphated or muriated waters in abdominal plethora and hepatic dyspepsia may also be suitably supplemented by thermal baths.

(*b*) *Disorders of the Blood.*—In anæmia, chlorosis, malaria, etc., baths act by promoting oxidation and change of tissue, by improving digestion and stimulating the nervous centres. In these conditions may often be remarked the restorative action, called by the French *reconstituant*.

(*c*) *Gout.*—The various nutritional disorders due to particular faults in the chemistry of digestion or to specific invasion, furnish a large majority of chronic invalids. If carefully regulated to the requirements of the case in hand, balneo-therapeutics afford to this large class an effectual means of treatment. In gout, the very common return, during spa treatment, of pain and tenderness, sometimes in long-forgotten sites of former attacks, indicates the searching influence both of waters and baths. In many cases no form of thermal bath is admissible, and the treatment is confined to the internal use of waters with dieting. In others, a suitable course of baths alone will effectually secure a long immunity from symptoms. This is equally the case whether the gouty manifestation be articular, or located in the digestive, circulatory, or nervous systems. The eliminative functions and the normal tissue exchange are alike restored.

(*d*) *Rheumatism.*—In joint affections, the result of rheumatic fever, as well as in those that gradually supervene on exposure to cold and damp, many forms of baths prove beneficial. In all recent and subacute articular changes, thermal treatment must be gentle and tentative; in chronic conditions more stimulating. For stiffness or adhesions, the result of bygone inflammation, a highly stimulating thermal treatment may be employed, and local applications made, of brines, peats, muds, vapour, and hot air in an ascending order of temperature. In chronic painful conditions of the fibrous tissues—the result of exposure or injury in predisposed subjects—thermal treatment and waters are of much service.

(*e*) (*Degenerative*) *arthritis.*—Most of the various forms of degenerative disease known as rheumatoid or osteo-arthritis are at one stage or another amenable to balneo-therapeutic treatment. It should, however, be carefully adapted to the stage and phase of the complaint. In the early, or inflammatory stage, which sometimes lasts over years, only a sedative, or subthermal bath is appropriate, *par excellence* the low-pressure massage-douche. Later, when inflammatory reaction has subsided and degenerative changes are marked, the blood supply of the joint is apt to be curtailed, and local thermal applications are beneficial by restoring the circulatory and nutritional activity. In the arthritis of middle life (a different clinical group) and in localised arthritis, the result of slight injuries in later life, balneary treatment may sometimes have the happiest results.

(*f*) *Senile atrophy.*—In atrophic changes, and in commencing fibroid degeneration, regulated thermal applications are of benefit by stimulating and equalising the arterial circulation and by bringing blood supply to ill-nourished parts. Subthermal warmth and moisture mollify the tissues and soothe and stimulate the nervous centres. In the old, high temperatures should only be locally applied.

(*g*) *Specific cachexias.*—Most or all cachexias, specific or otherwise, are more or less amenable to a properly devised health-resort treatment, with appropriate waters, baths, climate, etc. Under improved conditions of nutrition the specific cause often becomes less operative for longer or shorter periods, and it may be even wholly or partially eliminated.

4. DISORDERS OF THE NERVOUS SYSTEM.—Both local and general nervous disorders, not due to irremediable structural disease, are favourably affected by an appropriate course of baths. (*a*) *Peripheral affections.*—Affections of the peripheral nerves and nerve endings—neuralgia, or neuritis, whether due to gout, lead, alcohol, or other toxic influence—are often advantageously

treated at the mineral spas. In addition to general eliminative treatment thermal baths are often employed, and especially the peat bath, to which douches may be afterwards added.

(*b*) *Central affections.*—In cases of "spinal neurasthenia," and in the early stages of tabes dorsalis and other forms of spinal paralysis, especially in syphilitic subjects, spinal douches, subthermal and peat baths are commonly prescribed. With regard to purely functional disorders, it may be stated that conditions of general sluggishness or debility on the one hand, and of nervous erythism or excitement on the other, can be readily influenced by an appropriate stimulant, or sedative, bath treatment. The first group includes most cases of "hysterical" and other functional paralyses, for which a tonic and stimulant regimen is required. In the second group are many sufficiently serious conditions of irritability and hyper-sensitiveness of the nervous centres, for which a gentle and continuous sedative action is required.

5. RESULTS OF INFLAMMATION.—Special military establishments are provided at some of the French and Italian spas, where inflammatory thickenings, contractions, and adhesions resulting from wounds and injuries are removed by a course of thermal treatment. Increased tissue change, which may probably become more active in recently or partially organised tissues than in healthy parts, and the heightened power of absorption from stimulation of the general and lymphatic circulations, no doubt explain these results. In the same manner effusions and adhesions, the result of general or local (*e.g.* pelvic) peritonitis, or pleuritis, with the chronic congestion and other symptoms to which they give rise, are often successfully treated by balneo-therapeutic measures.

The author would here subjoin a series of RULES OF PRACTICE AND PRACTICAL OBSERVATIONS from his own experience with reference to the treatment of patients by baths:—

1. Before baths of any kind are employed an *examination of the cardio-vascular system* should be made, and a general view arrived at as to its structural and functional condition. The effect of the baths on the circulatory organs must be carefully watched throughout the treatment.

2. *Age and Sex.*—In children and in advanced life extreme temperatures should be almost always avoided. Baths at or near the point of thermal indifference are well borne in the old, and are often very beneficial. The same rule applies to women at the climacteric epoch. No mineral baths should be used during the menstrual period.

3. *The effect* of every bath, of whatever kind, is, in each particular case, either sedative or stimulant. The alternative will often depend on a very narrow range of temperature or duration, or even on the condition of the patient as affected by the time of day, the taking of rest, food, etc. It is therefore needful to discover and utilise the means by which the desired effect can be obtained, especially in cases of circulatory disorder.

4. *The duration* of Thermal baths and the length of the course must be regulated with care. They must never be so prolonged as to cause marked or persistent debility.

5. As it is the tendency both of mineral waters and baths to increase congestion or inflammatory reaction in affected tissues, *they must never be employed in febrile or inflammatory states.* The *primæ viæ* must be opened and visceral congestions and other acute derangements removed where necessary by preliminary treatment.

6. A certain *recrudescence of symptoms* in the affected parts is to be looked for as the natural effect of spa treatment. Within certain limits it is a

favourable sign. The slow chronic action is replaced by a more acute phase of disturbance. When this presently subsides under the continuance of treatment a healthier action follows. In this sequence lies a principle governing the treatment of all chronic maladies. If the recrudescence of symptoms should be early or severe the treatment must be modified or intermitted, and baths given less often or somewhat cooler (less stimulating). The reaction, marked by circulatory and nervous debility, that follows too prolonged or over-stimulating a course of baths is carefully to be avoided.

7. The amount of *physical exercise* permitted during the course of baths must be regulated for each case. In all cases of debility every exertion after the bath must be avoided, even that of dressing and the use of stairs. The sedative effect of baths in circulatory affections is frequently neutralised by subsequent fatigue. The patient should be comfortably wrapped and rest in a cool dressing-room. The hot pack (French *maillot*) must not be used where a sedative effect is desired.

8. The *after-treatment*, following immediately upon a course of baths, is a matter of consequence in many cases. In some a complete rest may be necessary, in all some degree of bracing is advisable. A week or two spent in some exhilarating climate among the hills or at the seaside, with pleasureable recreation, but without over-excitement or fatigue, should be prescribed in all cases, in which a course of treatment has been taken for serious organic or functional malady.

9. The *local employment* of balneary remedies has an important place in spa treatment. In many localised diseases (*e.g.* many cases of degenerative arthritis) a purely local thermal application is often beneficial when general thermal treatment is unnecessary or contra-indicated. The same applies to some conditions of debility, and to many cases in advanced life, for which a local application of heat may prove quite as stimulating as a general application would be for robust persons.

5. Summary of the Chief Mineral Waters and Baths

[*Note.*—In the following summary MT. signifies Mean Temperature and R. Rainfall, and the figures in italics represent the altitude in feet of the station above sea-level.]

Great Britain

Thermal and Subthermal Waters.—Bath (Somerset) *100*, R. 32, MT. 50·5°. The *Aquæ Sulis*, or *Solis*, of the Romans, an indifferent thermal water, temperature 104° to 120° F. The waters, which are but little used internally, contain calcium sulphate and carbonate, and small quantities of the chlorides of magnesium and sodium, in all 168 grains per gallon, with 4 grains of nitrogen gas. They are used in various thermal applications, including immersion baths and douches, and by pulverisation and natural vapour baths, general and local (Berthollet apparatus). Douches are combined with massage, as at Aix-les-Bains and Vichy. Buxton (Derbyshire) *1000*, R. 48·2, MT. 46·2°, a subthermal water, temperature 82° F. The waters, which are weakly mineralised (27 grains per gallon) contain bicarbonates of calcium and magnesium, with considerable quantities of free nitrogen. There is also a chalybeate water. Immersion baths and douches are used at both the natural and at higher temperatures, with and without massage. At the natural heat the baths produce a sedative effect. Taken altogether, the Buxton treatment, in which climate is an important factor, is well adapted to conditions of weakness the result of chronic disease. MATLOCK BATH (Derbyshire), indifferent water at 68° F. BAKEWELL (Derbyshire) *400*, indifferent water at 60° F. CLIFTON (Somerset) has a "hot well" at 73° F. MALLOW (County Cork), an indifferent water at 70°-72° F., the only warm spring in Ireland, and formerly much frequented.

Sulphur Waters.—HARROGATE (Yorkshire), *260-600*, R. 28·5, MT. 46·5°, a bracing climate with a comparatively dry atmosphere. Cold muriated-sulphur and chalybeate waters. The sulphur waters are numerous, and mostly impregnated with salt. Sodium sulphide occurs in them in quantities varying from 1 to 5 or more grains, and sodium chloride from 116 to 893 grains per gallon. In the different chalybeates the iron is found as carbonate, sulphate, and chloride. The waters are used externally and internally. There are also different forms of douche, with and without massage. STRATHPEFFER SPA (Ross-shire, Scotland) *200*, R. 31, MT. 45·5°, sulphur and chalybeate waters, peat and douche baths. The climate is sheltered, cool, and bracing. Excepting Laurvig and some others in Norway, Strathpeffer is the northernmost of European spas. Many sources of sulphur water, containing a relatively small amount of solids, chiefly sulphates of calcium and sodium, and sulphuretted hydrogen 3 to 19 cubic inches per gallon. The chalybeate well contains 2·46 grains of the carbonate of iron. In addition to the sulphur baths, a low-pressure douche-massage is employed at Strathpeffer. The peat baths are prepared from disintegrated peat or moor earth taken from deep beds on the side of Ben Wyvis. LLANDRINDOD (Radnorshire), *700*, R. 35, climate bracing and exhilarating, a cold muriated-sulphur and chalybeate spa. The amount of salt (163 to 334 grains) is considerably less than at Harrogate, and there is a relatively large amount of chloride of calcium (47 to 98 grains). The sulphur occurs as sulphuretted hydrogen (2 to 4 cubic inches). The chalybeate water is also muriated (chloride of sodium 278 grains) and contains 1·26 grains of ferrous carbonate to the gallon. The usual sulphur-water and douche baths supplement the internal use of the waters. LLANWRTYD (Brecknockshire) *800*, more sheltered than Harrogate, a pure sulphur water (chloride of sodium 60 grains, sulphuretted hydrogen 10 cubic inches). BUILTH (Brecknockshire) about *400*, a more strongly muriated-sulphur water (chloride of sodium 878 grains per gallon). MOFFAT (Dumfriesshire) *400*, mild sulphuretted and chalybeate waters. LISDOONVARNA (Co. Clare) *430*, sulphur and chalybeate waters. Among other sulphur waters in Great Britain may be mentioned: ASKERN SPA (near Doncaster), with 10 to 15 cubic inches of sulphuretted hydrogen; GILSLAND SPA (near Carlisle); SHAP WELLS (Westmoreland); CROFT SPA (Yorkshire); and DINSDALE-ON-TEES (Durham); also in Ireland, LUCAN (Co. Kildare); SWANLINBAR (Co. Cavan); and BALLYNAHINCH (Co. Down).

Muriated Waters.—DROITWICH (Worcestershire) *200*, R. 26·5, MT. 50·3.° The brine of Droitwich is one of the strongest known, for like the Dead Sea it is nearly saturated with common salt. It contains 21,761 grains per gallon, whilst sea water, the most familiar of muriated waters and salt baths, contains 1946 grains. Like other highly muriated waters that of Droitwich can only be used externally, either as baths, or lotions, or compresses, or by pulverisation to the mucous surfaces. The stimulating effect of these outward applications is sometimes very valuable, and the sense of warmth experienced after the bath illustrates this effect, produced through the vessels of the skin. NANTWICH (Cheshire) *150*, a somewhat similar brine to that of Droitwich, used for immersion baths. WOODHALL

SPA (Lincolnshire) *37*, R 22·7, a cold muriated spring, which may probably have a connection with the sea, containing 1330 grains of chloride of sodium, with 3 grains of bromides and a minute quantity of free iodine. The water is used for hot and tepid baths and compresses, and for pulverisation. There are also douches and douche-massage. CHELTENHAM (Gloucestershire) *150*, R. 29·8, MT. 47·1°, a muriated sulphated water. The stronger muriated waters contain 392 grains of chloride and 154 grains of sulphate of sodium, with very little of magnesium salts. There are also sources containing magnesium sulphate (126 grains) and about 100 grains of the carbonate and sulphate of calcium, and finally a carbonated chalybeate spring. LEAMINGTON (Warwickshire) about *200*, R. 30, MT. 48°, a muriated sulphated water, with a relatively large proportion of calcium sulphate. The large quantity of sodium chloride (678 grains) gives the water a slightly aperient action. There are excellent Turkish and other baths. BRIDGE-OF-ALLAN (Stirlingshire) *40*, climate sheltered; a muriated water containing about 380 grains of sodium chloride, and a relatively large quantity of calcium chloride. The water is taken internally and used for baths. LLANGAMMARCH (Brecknockshire) about *600*, has a bracing climate and a muriated water containing chlorides of sodium, magnesium, and barium (of the latter 6·7 grains), used both internally and for baths. ASHBY-DE-LA-ZOUCHE (Leicestershire) *400*, has a brine nearly equal in strength to sea water, at a natural temperature of 62° F.

CHALYBEATE WATERS.—TUNBRIDGE WELLS (Kent) *420*, a pure chalybeate, containing about 4·5 grains of the carbonate of iron, only used internally. Note also CHELTENHAM, HARROGATE, STRATHPEFFER, BUXTON. Many chalybeate wells in Great Britain, formerly much resorted to in their several localities, have now fallen into disuse. Among those that are at present employed is that of FLITWICK (Bedfordshire), which is bottled and sold. In this water the iron occurs in the unusual form of Ferric sulphate. The chalybeate at SANDROCK near Blackgang Chine, that of MOFFAT in Scotland, and at TREFRIW in Wales, besides others, contain the proto-sulphate of iron; and there are carbonated chalybeate waters in most parts of the country.

FRANCE

INDIFFERENT THERMAL WATERS.—PLOMBIÈRES, *1300*, in the Vosges mountains, temperature 77° to 155° F., natural vapour baths and inhalations, as well as douches and subthermal immersion baths. ST. AMAND, *100*, temperature 70° to 79° F., subthermal and sulphuretted mud baths. NERIS, *1150*, temperature 102° to 120° F. MONT DORE, *3440*, in the Auvergne mountains. The waters vary in temperature from 104° to 116° F. and contain a trace of arsenic. The baths are thermal, sometimes as high as 113° F., and very hot pediluvia are used. The water is applied to the respiratory passages by pulverisation and inhalation. DAX (near Bayonne), temperature 88° to 147°, thermal mud and water baths, douches, inhalations, etc. BAGNÈRES DE BIGORRE (Pyrenees) *1890*, thermal (77° to 123° F.) feebly sulphurous and chalybeate waters. In addition to ordinary thermal treatment, prolonged subthermal baths are given. AIX-LES-BAINS (Savoy) *860*. The thermal waters (109° to 112° F.) contain a trace of sulphuretted hydrogen. The neighbouring water of Challes, a very strong sulphur water, is employed for internal use; and that of Marlioz, in close proximity to Aix, for inhalation, etc. The characteristic treatment at Aix is the massage-douche in its various applications. The waters are also used for local and general vapour baths.

Muriated Waters.—The following are thermal:—CHATEL-GUYON, *1300*, in the Auvergne mountains; BOURBON L'ARCHAMBAULT, *870*, and BOURBON LANCY, *780*, (both near Moulins); BOURBONNE-LES-BAINS (Dep. of Haute Marne) *900*; LA-MOTTE-LES-BAINS (Isère) *2000*, and BALARUC, (near Cette). Salins *1200*, in the Jura mountains, possesses a cold muriated water with traces of bromides and iodides.

Alkaline Waters.—VICHY (Dep. of Allier), *736*, cold and warm springs, containing bicarbonate of sodium (more than three hundred grains per gallon) with free carbonic acid and, in one case, a small quantity of iron. Inhalation, douche, and other baths. VALS (Dep. of Ardèche) a cold alkaline spring, rich in carbonic acid gas. ROYAT, *1480*, has subthermal muriated alkaline water, containing bicarbonate of iron and free carbonic acid gas. The baths are subthermal or effervescing and carbonic acid gas is also applied locally and generally. The waters of SAINT NECTAIRE, *2500*, are similar. LA BOURBOULE, *2780*, has a thermal muriated alkaline water containing arseniate of sodium, and used for drinking as well as for baths and inhalations. Like the two preceding spas it is situated in the Auvergne mountains. EVIAN-LES-BAINS, *1240*, on the Lake of Geneva, possesses a feebly alkaline water containing carbonate of calcium.

Sulphated Muriated Waters.—BRIDES-LES-BAINS and SALINS-MOUTIER (Savoy), *16-1800*, have subthermal waters containing common salt and the sulphates of sodium, magnesium, and calcium—with carbonated and other baths. SAINT GERVAIS, *2000*, near Chamounix, has similar waters.

Calcareous Waters.—CONTREXÉVILLE (Dep. of Vosges), *1150*. The water is cold and contains sulphurate and bicarbonate of lime. POUGES-LES-EAUX (Dep. of Nièvre), *650*; CRANSAC (near the Volcano le Montet), *980*, and having also natural sulphurous vapour baths; MARTIGNY-LES-BAINS and VITTEL, *11-1200* (near Contrexéville), possess similar waters.

Chalybeate Waters.—LAMALOU, *620*, in the Cevennes mountains, has thermal and weakly alkaline carbonated chalybeate water, and subthermal "piscine" baths. RENNES-LES-BAINS (Dep. of Aude), *1040*, carbonate and sulphate of iron springs. OREZZA (Corsica) has gaseous chalybeate waters. See also Indifferent Waters, BAGNERES DE BIGORRE.

Sulphur Waters.—The following are thermal, and situated in the Pyrenees. They all contain sulphur in the form of sodium sulphide or sulphydrate in proportions varying from 1 to 7 parts in 100,000. BAGNÈRES DE LUCHON, *2000*. The waters (sulphide 5 to 7 parts per 100,000) are used internally and for baths, inhalations, etc.; CAUTERETS, *3200* (sulphide 1 to 2 parts); EAUX BONNES, *2400* (sulphide 2 parts); EAUX CHAUDES, *2050* (sulphide 0·8 part). BARÈGES, *4200*, (sulphide 4 parts), has highly stimulant thermal baths and douches, the effects of which are aided by climate. The "baregine" or organic matter of the springs consists of an alga (*Leptomitus sulphurarius*) which is peculiar to thermal sulphur waters. ST. SAUVEUR, *2500* (sulphide 2 parts), sedative and subthermal baths. AX-LES-THERMES, *2340* (sulphide 1 to 2 parts), AMELIE-LES-BAINS, *920* (sulphide 1 part) and VERNET-LES-BAINS, *2060* (sulphide 4 parts). URIAGE, *1350*, is a subthermal spring in the Dauphiné Alps, containing sulphuretted hydrogen. CHALLES (Savoy), *880*, possesses one of the strongest cold sulphur waters in Europe. The sulphur occurs as sodium sulphide (50 parts per 100,000), together with iodide and bromide. This water is much employed locally for the respiratory mucous membranes (pulverisations, inhalations, etc.) as well as internally (see Indifferent Thermal Waters, AIX LES BAINS). PIETRAPOLA and GUAGNO (Corsica) are thermal sulphur springs containing about 2 parts of sulphide.

GERMANY

INDIFFERENT THERMAL WATERS.—WILDBAD, *1410*, in the Black Forest, temperature 91° to 104° F., subthermal and vapour baths and douches. SCHLANGENBAD (Nassau), *900*. The water (82° to 90° F.) contains nitrogen and oxygen gas, and is used for subthermal baths, which have a valuable alterative and sedative quality. The water of BADENWEILER, *1400*, also in the Black Forest, has a temperature of 79° F. LANDECK and WARMBRUNN (in Prussian Silesia) are subthermal waters containing minute quantities of sulphur.

MURIATED WATERS.—NAUHEIM (Grand Duchy of Hesse), *400*, a subthermal (82° to 95·5° F.) muriated water with 1 to 3 per cent of salt, and rich in free carbonic acid gas. There is also a cold chalybeate. The baths subthermal, muriated or muriated and gaseous. There are also carbonic acid gas baths. KREUZNACH (Rhenish Prussia), *340*. The waters are cold and contain chloride of sodium 1 per cent, and chloride of calcium 0·1 per cent. Douches, pulverisations, hot air baths, etc. HOMBURG (Hesse-Nassau), *600*, has cold muriated chalybeate springs, effervescent with carbonic acid gas. WIESBADEN (in the same province), *380*, possesses muriated waters at 100° to 156° F., with a considerable gaseous element. The thermal baths are highly stimulating. Douches, vapour baths, etc. KISSINGEN (Bavaria), *600*, a cold muriated water, containing much free carbonic acid gas and a small quantity of iron. Besides hot salt baths, mud baths, and inhalations, gas baths are here employed. BADEN-BADEN, *650*, numerous thermal muriated springs (124° to 150° F.), with traces of lithium and arsenic. Baths of various kinds, including thermal, hot air, vapour, and local mud baths. OEYNHAUSEN (Westphalia), *230*, and SODEN (Hesse-Nassau), *450*, have subthermal carbonated muriated waters resembling those of Nauheim. See also Sulphur Waters—AACHEN, and Chalybeate Waters —PYRMONT.

ALKALINE WATERS.—NEUENAHR (Rhenish Prussia), *760*, has subthermal (96° F.) alkaline waters, employed for drinking, baths, and inhalations. SALZBRUNN (Prussian Silesia), *1320*, has cold alkaline waters. At EMS (Prussia), *300*, there are numerous thermal (80° to 120° F.) alkaline muriated springs, much used internally and by inhalation for catarrhal conditions. ELSTER (Saxony), *1500*, has a cold sulphated alkaline water containing much free carbonic acid gas. There is an effervescing chalybeate which is used for baths and there are also moor baths.

CALCAREOUS WATERS.—WILDUNGEN (Waldeck), *980*, possesses cold carbonated waters (calcium and magnesium), with much free carbonic acid gas. LIPPSPRINGE (Westphalia), *450*, is a weak calcareous water at 70° F., with some nitrogen gas.

CHALYBEATE WATERS.—SCHWALBACH, *950* (in the Taunus range), has a strong cold carbonated effervescent chalybeate water, much used internally and for baths. Peat baths are also given. At KUDOWA (Silesia), *1270*, there is an alkaline chalybeate, with peat and gas baths. DRIBURG (Westphalia) and RIPPOLDSAU and other health resorts in the Black Forest possess valuable carbonated chalybeate waters. PYRMONT, *420*, has both muriated and effervescing chalybeate waters, and provides gaseous-iron and moor baths.

SULPHUR WATERS.—AACHEN (AIX-LA-CHAPELLE), *530*. The thermal waters (113° to 133° F.) are muriated and sulphurous, and are used for drinking, baths, and inhalations. The baths are much employed as an adjunct to the mercurial treatment of syphilis. At WEILBACH (Hesse-

Nassau), *440*, the sulphur springs are cold and alkaline. NENNDORF in the same province has very strong sulphuretted waters and gaseous and sulphurous mud baths. At MEINBERG (Lippe Detmold) and EILSEN there are also strong cold sulphuretted and muriated springs with similar baths.

HOLLAND AND BELGIUM

INDIFFERENT THERMAL WATER.—CHAUDFONTAINE, near Liège, temperature 96° F.

CHALYBEATE WATERS.—SPA, *1000*, in the Ardennes, a cold sparkling carbonated water, gaseous chalybeate, douche, and moor baths. At HAARLEM (Holland) there is a cold muriated chalybeate water.

SWITZERLAND

INDIFFERENT THERMAL WATERS.—RAGATZ-PFAEFERS, *1700*, a subthermal (98·6° F.) water rich in nitrogen gas, used for ordinary and prolonged baths. LOËCHE-LES-BAINS (LEUK), *4600*. The temperature of the waters ranges from 102° to 124°. Baths of several hours' duration are taken, in "piscinæ" and otherwise, at temperatures of 93° to 95°.

MURIATED WATERS.—RHEINFELDEN, about *850*, has a nearly saturated brine, used for immersion baths and douches. The brines of SCHWEIZERHALLE and BEX are somewhat similar.

ALKALINE WATERS.—PASSUGG (near Chur), *2710*, possesses an acidulous alkaline spring similar to the "Grande Grille" at Vichy. At TARASP-SCHULS (Canton Grisons), *3890*, the water is also alkaline and effervescing, and contains sulphate as well as bicarbonate of sodium. There are, moreover, alkaline chalybeate waters and gaseous and mud baths.

CHALYBEATE WATERS.—The waters of ST. MORITZ in the Upper Engadine, *5800*, are weakly chalybeate and effervescing, and used internally and for baths. ACQUAROSSA (Canton Ticino), *1150*, has a warm chalybeate spring. That of SAN BERNARDINO (Grisons), *5380*, is cold and gaseous.

SULPHUR WATERS.—BADEN (Canton Aargau), *1230*, has a weak thermal sulphur water at 118° F. SCHINZNACH in the same Canton, *1140*, possesses a very strong subthermal sulphur water (82° to 95° F.) Prolonged and ordinary baths and inhalations are used. LAVEY and YVERDON (Canton Vaud) yield weak thermal waters of the same class. LENK (Canton Bern), *3600*, has a cold and strong sulphuretted water used for drinking and pulverisation; and a similar water is met with at GURNIGEL, *3780*, in the same Canton.

CALCAREOUS WATER.—That of WEISSENBURG (Canton of Bern), *2828*, has a temperature of 79° F., and contains a small quantity of sulphate of calcium and magnesium.

AUSTRIA

INDIFFERENT THERMAL WATERS.—GASTEIN, *3310*, is one of the highest baths in Europe and a typical member of its class. The waters range in temperature from 78° to 121° F. TEPLITZ (Bohemia), *730*, has a slightly alkaline thermal water (temperature 83° to 114° F.), and peat baths. JOHANNISBAD, *2300*, temperature 85° F.

MURIATED WATERS.—At ISCHL, *1500*, there is a strong brine with weaker waters for internal use, and moor and other baths. HERCULESBAD (in Hungary) possesses a thermal muriated water.

ALKALINE WATERS.—There is a strong and gaseous alkaline water at

BILIN in Bohemia. That of GLEICHENBURG (Styria), *930*, is a very similar water, but contains chloride of sodium. KARLSBAD (in Bohemia), *1230*, has sources of thermal water (from 95° to 162° F.), which are both alkaline and sulphated, with bicarbonate and sulphate of sodium. These celebrated springs are much used internally, and moor, thermal, douche, vapour, and other baths are also employed. At the neighbour spa, MARIENBAD, *2000*, there are similar but cold waters, as well as chalybeate springs, and peat, chalybeate, and gaseous baths. FRANZENSBAD, also in Bohemia, *1500*, possesses cold effervescing springs of the same class, as well as chalybeates. This spa is also the home of the peat or moor bath, which is employed both locally and generally.

SULPHATED WATERS.—Hungary is rich in these "bitter waters." FRANZ-JOSEPH, HUNYADI-JANOS, APENTA, and ÆSCULAP are rich in sulphates of magnesium and sodium. PULLNA and SEIDLITZ, in Bohemia, are weaker waters of the same class, the latter containing no sodium salts.

CHALYBEATE AND ARSENICAL WATERS.—See Alkaline Waters—FRANZENSBAD and MARIENBAD. RATZES and MITTERBAD, in the Austrian Tyrol, *3-4000*, have sulphate of iron springs, and LEVICO and RONCEGNO in the same region yield strong ferruginous waters containing arsenious acid, which are used at the source and for exportation.

ITALY

INDIFFERENT THERMAL WATERS.—The thermal sources of Italy are very numerous, and were many of them used by the Romans. BORMIO (N. Italy), *4500*, 91° to 105° F., and BATTAGLIA (Province of Venice), *1900*, are indifferent waters. The latter has sweating grottoes of natural vapour and mud baths. MONSUMMANO has also a natural vapour bath.

MURIATED WATERS.—ABANO (Padua), *100*, has hot (100° to 183° F.) muriated waters and mud baths. SALSOMAGGIORE (Parma), *520*, yields a cold muriated water and a carbonated chalybeate. MONTECATINI (Lucca), *920*, is described as one of the first balneary stations in Italy. The waters are subthermal and weakly muriated.

CHALYBEATE AND ARSENICAL WATERS.—The hot spring at PISCIARELLI (near Pozzuoli) contains sulphate of iron, and CERESOLE REALE (Piedmont), *5290*, has carbonated iron waters with arseniate of sodium.

MURIATED ALKALINE WATERS.—At Pozzuoli (Puteoli), on the Bay of Naples, there are thermal, alkaline, and weakly muriated waters that have been used for baths since Roman times. The famous *solfatara*, an ancient crater, was formerly used for vapour baths. In the crevices of the hills are numerous *fumeroles* giving forth sulphuretted hydrogen. Vapour baths, *stufe*, are given at 95° and 113° F. The neighbouring thermal spring at BAGNOLI is somewhat similar, and furnishes vapour for the *Bagni di Nerone*. CASAMICCIOLA (Ischia) has also a thermal spring of this class. The waters of CASTELLAMARI DI STABIA (Bay of Naples) are cold, and include alkaline, muriated, and ferruginous sources.

SULPHUR WATERS.—ACQUI (near Alessandria), *450*, one of the chief Italian spas, has a hot muriated sulphur water (102° to 158° F.). Thermal and mineral mud baths are used. Other hot sulphur springs are VINADIO (Piedmont), PORETTA (Bologna), *1100*, and VITERBO, ACQUE ALBULE and CIVITTA VECCHIA in the province of Rome; also ACIREALE and SCIACCA in Sicily.

CALCAREOUS WATERS.—LUCCA, *390*, is an ancient and famous bath, supplied by thermal, earthy, and muriated waters at 98° to 129° F. CHIANCIANO in central Italy is also a thermal calcareous water.

GREECE

Greece is well supplied with thermal and other mineral waters. Though much resorted to in ancient times, they are now almost entirely neglected. Among the more important sulphurous thermal springs may be named HELLOPIA in Epirus, ADEPSOS (I. of Eubea), those of the island of LESBOS, and at THERMOPYLÆ. LEPANTO, and KATHARSION (in Lesbos) are saline sulphur waters. The islands of the Ægean Sea furnish many powerful waters, for example the thermal waters of IKARIA and KYTHMOS. There are bitter waters at EPIDAURUS.

SPAIN AND PORTUGAL

FITERO (province of Navarra) and CALDAS-DE-GEREZ (Portugal), a feebly sulphurous thermal water at 115° F., are practically indifferent thermal waters. There are several hot muriated waters in Spain, the most important being that of CALDAS-DE-MONTBUY (Barcelona). VIDAGO (N. Portugal) has a strongly alkaline water. RUBINAT on the French frontier yields the bitter sulphated waters called "Rubinat" and "Condal." Among sulphur springs that of PANTICOSA in the Pyrenees, *5600*, is a thermal water resembling some of those on the French side of the mountains, and containing two parts of sulphide of sodium in 100,000. At such an altitude, almost the highest of European spas, bath treatment is greatly modified by climate. CARRATRACA (near Malaga), MONTEMAYOR Y BEJAR and LEDESMA, *2600* (both near Salamanca), and CALDAS DA RAINHA (Portugal), are among many other sulphur springs. URBEROAGO DE ALZOLA (near Plasenzia) has warm calcareous and chalybeate springs.

NORWAY, SWEDEN, AND ICELAND

SANDFJORD and LAURVIK (near Christiania) possess both sulphurous and chalybeate springs, and employ baths and frictions with sulphurous mud. Mud baths are also much used at MODUM, where there is also an effervescing chalybeate water (ST. OLAFS). Among the numerous chalybeate waters of Sweden may be mentioned MEDEWI, DANEVERD (near Upsala), and GUSTAFSBERG (near Uddevalla). At the two last mud baths are commonly used, also at WAHLBERGA (near the Lake of Wenver), and at LOKA (on Lake Lersjö). In Iceland, BARESTRAND SYSSELL has indifferent waters at 218° F., but RANDAMEL, with ferruginous waters, is more frequented.

RUSSIA

Among the better-known Russian spas are KÖTSCHENOWA (near Moscow), an effervescing chalybeate water; STOLYPIN (on the Kuschum), a saline sulphur water; in Poland, CIECHOCINCK, with strongly muriated springs, and BUSK (near Cracow), with muriated sulphur waters and peat baths; on the Baltic, OESEL (in Livonia), with sea and mineral mud baths, and KEMMERN (near Riga), with cold sulphur waters and peat and sea baths. There is in the Caucasus, at PJÄTIGORSK and its neighbourhood, an important group of thermal waters, some sulphurous and others ferruginous or alkaline. One of these, SCHELNESA-WODSK, *1800*, is an alkaline ferruginous spa and much frequented. Among other Caucasian springs, BORGUM has strong sulphurous alkaline waters. Bituminous and brine mud baths are given at ASTRAKHAN on the Caspian Sea, and sea mud baths are much made use of at ODESSA and on the Black Sea coast.[1]

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[1] For many of the particulars with reference to the European spas, the author desires to express his indebtedness to the admirable work on the *Mineral Waters and Health Resorts of Europe*, by Sir Hermann and Dr. Parkes Weber, and to Bradshaw's *Dictionary of Mineral Waters*.

TURKEY, BULGARIA, etc.

There are some very complete thermal baths in Turkey. BRUSSA (Asia Minor), with many thermal springs, is said to have one of the finest balneary establishments in the world. Among many others, there are thermal muriated and ferruginous waters at AHIOLO (near Constantinople), and thermal sulphur waters at KIRKILISSA (Bulgaria), at SMYRNA, at CALLIRRHOË (Palestine), and at SALONICHI (Macedonia), where mud baths are also used. Other thermal waters are found at FEREDSCHIK (Roumelia); LIDJA (Anatolia), and GADARA (Palestine), where sulphurous and mud baths at high temperatures are used by the Arabs.

AFRICA

HELOUAN-LES-BAINS on the desert near Cairo, *200*, a remarkably dry winter climate. Muriated and muriated sulphur springs at 90° F. Inhalations. Douche and other baths.

Algiers has many valuable thermal spas. The water of BAINS-DE-LA-REINE (near Oran) is muriated, at 130° F., as also that of HAMMAM MELOUAN. HAMMAN R'IRHA has hot calcareous and chalybeate waters, and HAMMAM MESKUTIN a hyperthermal water at 203° F. MARA (Abyssinia) has bitter waters, and OKME (Nubia) warm sulphur springs.

INDIA

As in all volcanic countries, so in India, mineral springs abound, but very few of them have at present been subjected to scientific investigation and analysis. Those in particular that occur at high altitudes might probably prove valuable to Europeans.

THERMAL SPRINGS.—Of this class there is a large number in India, especially in the TENASSERIM provinces. In the HAZARIBAGH district (Bengal), the country, for more than 100 miles in all directions, is teeming with thermal springs, at an altitude of about 1500 feet. At MUNNIKARN (Jalandhar Doab) there is a boiling fountain (207° F.) at an elevation of 5587 feet, as well as waters containing iodine and bromine. There are also many in the HIMALAYAS and in SINDH.

SULPHUROUS THERMAL WATERS are also numerous in India. Among the better-known are:—JAWALA MUKHI ("Flames mouth"), in the Kangra district of the Punjaub. There are here many sources, some of a remarkable character, containing iodine as well as sulphur. They are used especially for goitre, from which the local inhabitants are said to be exempt. Combustible gas issues in jets, which are kept constantly burning as a manifestation of the Hindu goddess Devi. There is also a sacred sulphurous spring at DANERA. At SONA (near Delhi), there are sulphur waters with a considerable establishment; also in the HAZARIBAGH district, and at high elevations in the Jalandhar Doab (BESHISHT, *6681*); also at DARJILING, at CANNEA (Ceylon), at MALACCA, and elsewhere. MURIATED springs are universal through the SALT RANGE (Punjaub), and occur in Western SINDH. CHALYBEATE waters are found in parts of the Himalaya and in the Neilgherries, and at NAGCONDA in the Simla hills.

AMERICA AND CANADA

ALKALINE WATERS.—Among Californian springs, the OJO CALIENTE is a thermal (100° F.) and weakly muriated alkaline water. The SARATOGA "VICHY" (New York State) is a somewhat similar source, but cold.

Muriated Waters.—There are many brines and muriated springs in America, artesian and other. At Saratoga, the Hawthorn and some other sources are of this kind; also Blue Lick Springs (Kentucky), and Saint Catharine's Well and the Caledonia Springs in Ontario, among others Canadian.

Sulphated or Bitter Waters are comparatively scarce in America. That of Crab Orchard (Kentucky) is one of the best-known.

Sulphuretted Waters are numerous in the States of New York and Virginia. Sulphurous inhalations are used at Richfield (N.Y.). The Salt Sulphur Spring (West Virginia), the Paroquet Spring (Kentucky), the Sharon and White Sulphur Springs (in the State of New York), and the Sandwich Spring (in Ontario), are among the best-known in the North American continent.

New Zealand

The most interesting mineral springs of New Zealand occur in the province of Auckland. Tarawera is a thermal water at 130° F. At Taupo, *1500*, there is an important group of hot springs. (1) The *Crow's Nest* spring is muriated and has a temperature of 170° F; (2) The *Ruahine* is still hotter, 190° F., and contains some free acid; (3) Thermal sulphurous; and (4) Alkaline siliceous waters. Te Aroha, *350*, has sulphurous muriated, alkaline, and effervescing carbonated waters. Ohinemutu, *962*, is situated on the southern shore of Lake Rotorua, the centre of the hot spring district. Among the chief sources are:—(1) *The Priest's Bath*, which is described as an acid, sulphurous water containing alum, at 98° to 106° F. There are here piscinæ, which are said to form "the finest bath in the southern hemisphere." (2) *Rachel's Bath*, a soft saline water at 194° F. (3) *The Blue Bath*, containing "salines and silicates," at 140° F. Mud baths are also given. It was in the neighbourhood of Ohinemutu that the famous "pink and white terraces" were destroyed by volcanic eruption in 1886.

Japan

Sulphurous Thermal Waters.—Kusatsu, *3500*, is the most celebrated of the Japanese spas. The waters are hyperthermal, and contain much sulphur, and also (it is said) arsenic and copper. The temperature of the "Fever Bath" is 150° F., that of the "Eagle Bath" somewhat cooler. A remarkable feature is the amount of free sulphuric acid in some of the baths. They are much used, and at very high temperatures, for syphilis, leprosy, and severe forms of rheumatism. Ashinoya (near Lake Hakone) possesses many hot sulphur springs and solfataras. It is frequented by the Japanese for skin affections and syphilis. Yumoto, *5061*, and Nikko (in the same district) have similar waters. Unzen, near Nagasaki, is also a sulphur spring.

Among *Chalybeate Waters* are Ikao, *2700*, "a cool and pleasant" resort near Nagasaki; and Arima (near Kobe), with hot iron springs and baths. The best-known of the Salt Springs of Japan are Atami and Isobe.

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Bandages: Varieties and Method of Application.

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Bandages vary in their make, quality, and method of application, to suit the purpose they are required to serve. They are employed to keep in place splints, dressings, and surgical appliances; to exercise uniform pressure upon a part, as in the sprains and the inflammation of joints; or to support the circulation, as in the treatment of varicose veins.

The different varieties of bandage may be grouped under the headings of triangular bandages, roller bandages, many-tailed bandages, and shaped bandages.

The Triangular Bandage is the simplest, and is often known as Mayor's or Esmarch's, the first aid or the military bandage, since it is chiefly used on field service to keep the temporary dressings in place. It is made by cutting diagonally across a square metre of linen or calico. It is used in three forms, either unfolded, when it is known as "the whole cloth," folded, so that the point or apex of the triangle is carried to the centre of the lower border, and then folded again in the same direction—

the "broad bandage"; or the broad bandage is again folded lengthwise to make the "narrow bandage." The whole cloth is very useful as a sling for the entire arm, and it is also employed to keep dressings in place in wounds of the head, chest, back, hands, or feet. The broad-fold bandage is a good sling when it is only necessary to support the forearm or hand, and is serviceable in the treatment of broken ribs; whilst the narrow-fold bandage is used to keep splints in place in the treatment of fractures. The bandage is fastened off by knotting its ends, reef-knots being always employed, and pins used as seldom as possible.

Roller Bandages.—These are employed in civil surgery as well as in all military hospitals. They consist of tightly rolled and continuous strips without seam or selvage, measuring from three to eight yards in length, and from three-quarters of an inch to three inches in width. They are made of calico, linen, flannel, domette, crinoline, gauze, or rubber. Calico and linen were alone employed until the introduction into surgery of antiseptic methods replaced them, in many instances, by strips of medicated or sterilised gauze. Calico or domette bandages are still used to keep splints in place, but their neat and effective application can only be obtained after long practice. The older surgeons brought the art of bandaging to a high pitch of perfection; thus Ambrose Paré says, "after putting on a bandage the surgeon should look to see that, if it be featly applied, it looks pretty, so as to satisfy both the patient and his own assistants, for each in his profession should beautify his work so far as in him lies." This wholesome counsel has been much neglected of late, and bandages are often applied in so slovenly a fashion as to be almost ineffective.

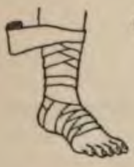
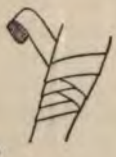


Fig. 1.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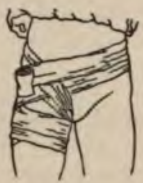
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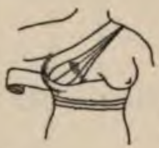
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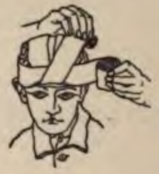
Fig. 5.

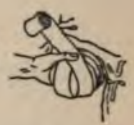
Fig. 6.

The simplest manner of applying a calico bandage to a limb is to wind it spirally from the extremity towards the trunk; but such a spiral bandage does not exert a uniform pressure unless the limb be of the same size throughout, and it soon becomes loose. A spiral bandage is therefore rarely applied, its place being taken by a "reverse" or a "figure-of-eight" bandage. The bandage by reverses (Fig. 1) is particularly adapted for those parts which increase in size gradually from below upwards, like the arm and leg, whilst the bandage by figure of eight (Fig. 2) is used when the enlargement of the limb is too great or too irregular for the reverses to lie evenly, as over the knee, the ankle-joint, and the elbow.

The hip and shoulder are enveloped in a *spica bandage*, so called from the resemblance of the pattern in the completed bandage to a spathe or ear of corn. It is applied to the hip (Fig. 3) by carrying the bandage round the upper part of the thigh and then obliquely across the abdomen, so that it passes between the great trochanter and the crest of the ilium on the opposite side. It is then carried round the back below the crest of the ilium, over the injured groin from the outer towards the inner side, round the thigh, and then over the lower part of the abdomen, taking care that each turn rises higher than the one previously applied. This—the ascending groin spica—is one of the most useful bandages, and is employed after hernia operations, and temporarily in place of a truss. In the shoulder the spica is applied by taking a turn of the bandage round the arm close to the axilla. It is then brought round the back, under the opposite armpit, across the front of the chest, over the affected shoulder, then sharply round the injured armpit, and so round the body again. The highest turn of this bandage is placed as near the root of the neck as possible, and each turn is carried lower than the preceding one, but still overlapping it in part, until the whole shoulder is covered, and the bandage is therefore a descending spica. Similar bandages are applied to the thumb and to the great toe, beginning at the wrist and ankle respectively.

The breast (Fig. 4) is supported, or dressings are kept in place upon it, by carrying a roller bandage once round the body below the breast, beginning in front and passing round the sound side. This circular turn secures the end of the bandage, and the roller is then carried obliquely over the lower part of the diseased breast to the opposite shoulder, which it crosses, and is then brought back beneath the axilla of the injured side to the circular turn. This is repeated, carrying each turn of the bandage a little higher than the preceding one until the breast is completely covered.

The Capelline Bandage (Fig. 5) is sometimes useful to keep dressings upon the head, though the triangular head bandage is usually the more convenient, because it is lighter and more easily applied. A double-headed roller is first made by rolling an ordinary bandage from opposite ends in such a manner that the two rolls meet when one is a third larger than the other. The operator then stands behind and on a higher level than the patient, and lays the centre of the double-headed roller as near the brows as possible. He then carries the two ends of the roller in opposite directions round the head until they meet below the occipital protuberance. The longer bandage is carried round the head again, whilst the shorter part is bent over the circular turn, which thus keeps it in place, and is carried forward to the brow, to be again caught by the circular turn. These manœuvres are repeated until the whole head is covered by the bandage, when the two ends are crossed, taken once or twice round the head just above the ears and in opposite directions, until they are tied together in the middle of the forehead.

Eye Bandages are usually made of tightly rolled strips of mull muslin 6 yards in length by 2½ inches in width. When one eye requires bandaging a pad of soft linen or absorbent wool is placed over the orbit, and the end of the roller bandage is placed on the side of the head just above and behind the ear of the sound side. The bandage is then carried round the head, so that it lies low upon the forehead in front and below the occipital protuberance behind. When the circuit has been completed the bandage is carried forwards a second time until it reaches the brow on the uninjured side. It is then reversed upwards to prevent the lower edge impeding the sight of the sound eye, and is carried obliquely downwards and forwards across the pad, below the ear on the injured side and below the occiput, until it joins the circular portion just at the point where the bandage was begun. A safety-pin is inserted at this point to keep it secure, and the bandage is then reversed and carried across the head to be pinned or tied to the circular portion of the bandage just behind the ear on the injured side.

The stump of a Limb (Fig. 6) is bandaged by encircling it a few times with a roller bandage, each turn of the bandage being nearer the end than the one before it. The roller is then brought forwards from the back of the stump over its face to the front, and it is then looped to and fro over the end of the stump until the whole surface is covered, the entire series of loops above and below being held in position until they can be secured by carrying the bandage circularly round the stump in such a manner as to bandage over the loops and so prevent them from slipping. This bandage not only keeps the dressings in place, but it supports the flaps and counteracts the tendency shown by the muscles to drag themselves away from the cut end of the bone.

Gauze Bandages are rollers made of strips of fine unbleached tarlatan, either sterilised or impregnated with some of the various antiseptics used in modern surgery. Carbolic gauze bandages are made by sterilising the muslin by heat after it has been washed and dried. It is then passed through a melted mixture consisting of four parts of paraffin, four parts of resin, and one part of pure carbolic acid; the excess of the mixture is squeezed out, and when the gauze is dry it should contain 5 per cent of carbolic acid in its meshes. The resin is employed to prevent the acid being washed out too quickly when it is brought into contact with a discharging wound, whilst the paraffin is used to diminish the stickiness of the resin. Iodoform gauze bandages should contain not less than 20 per cent of iodoform. They are made by rubbing the powder into dehydrated and sterilised gauze. A little glycerine is often added to fix the iodoform, and to render the gauze more supple. The blue sal-alembroth (a double chloride of mercury and ammonium) gauze bandages contain 1 per cent of the salt, but they are so irritating to the skin that I have given up using them even for adults. Gauze bandages have the advantage of being very light and comfortable to wear: they have the disadvantage of tearing easily and of sticking very tightly to the skin.

THE CHIEF "MADE" BANDAGES are the many-tailed bandages, often known as the bandages of Scultetus (1595-1645). The four-tailed bandage used in the treatment of fractured jaw was for a long time the only form of this type of bandage used in England, though the many-tailed bandage in its different varieties has always been a favourite with the French surgeons, but of late years it has been more widely used in this country. Scultetus' bandage is made by cutting a roller bandage into a sufficient number of pieces to cover the part, each piece being long enough to go round the body or limb and overlap two or three inches. The strips are then arranged parallel to each other, so that each overlaps the one above it, and the whole series is slipped under the wounded part. The ends of each strip are then brought round separately, and are made to fold over each other, a sufficient number of safety-pins being inserted to keep them all secure. It is the especial merit of this form of bandage that a soiled portion can be removed easily and without disturbing the whole bandage, a merit which is counteracted by the modified form in which all the pieces are held together by stitching the centre of each strip to a piece of bandage running vertically. The many-tailed bandage is particularly serviceable when it is desired to move the patient as little as possible after a severe operation, as after an abdominal section, herniotomy, or amputation of the breast.

A *T-Bandage* is used to keep dressings on the perinæum or on the head. It consists of two strips of roller bandage, one piece being long enough to pass once or twice round the waist or head, the other a longitudinal piece sewed at right angles to the transverse piece and at its centre. The longitudinal piece is half the length of the transverse piece, and when two longitudinal strips are used a double T-bandage is formed. Many useful varieties of a made bandage can be invented by sewing a strap and buckle to pieces of gauze or lint shaped to cover the injured part.

Fixed Bandages. — Calico and roller bandages may be prevented from shifting by rubbing a solution of starch paste over them after they have been applied in the manner desired. The starch paste is made by boiling starch in water until it is quite thin, and it is then applied whilst it is hot by rubbing it over the bandage or by painting it on with a brush. The bandage sometimes shrinks if the paste has been too freely applied, and it should therefore be watched to see that it does not become unduly tight. Bandages can also be stiffened by impregnating them with a mixture of gum and chalk (now obsolete), by soaking them in melted paraffin, and by painting them over with a saturated solution of silicate of soda. Plaster of Paris, however, has practically replaced these different methods, though it is so difficult to remove that I sometimes prefer silicate bandages, as they soften easily when they are soaked in water. Plaster of Paris bandages are prepared by rubbing freshly-baked gypsum into crinoline or some other variety of loose-webbed bandage. The bandage is then loosely rolled, and is placed in a basin of water until bubbles cease to come from it. The part to be bandaged is protected by a flannel bandage, over which the wet plaster bandage is applied. Care must be taken not to bandage too tightly, and the part must be held in the required position until the bandage has set. The setting can be hastened to any required extent by adding more or less potassium sulphate to the water in which the bandage is soaked. More than one thickness of bandage is required to get a sufficiently rigid case, and the bandage is applied spirally or in a figure of eight, never by reverses. It is removed by soaking or by cutting through the bandage either with a saw, scissors, or specially-designed cutting forceps. Plaster of Paris bandages are used more especially in the treatment of talipes, either with or without tenotomy, after osteotomy and excision, in some forms of simple fracture, and as a jacket in spinal caries.

Rubber Bandages.—These are used to control or to regulate the circulation through a limb in the two forms of Esmarch's and Martin's bandages. An *Esmarch's bandage* is employed to arrest the circulation through a part in cases of severe hæmorrhage or to render a surgical operation bloodless. It consists of a long strip of very stout indiarubber, wound like an ordinary roller bandage, which is applied to the limb from below upwards to the spot where the blood-flow is to be stopped. A piece of strong indiarubber tubing is passed round the limb just above the upper border of the bandage, and is pulled tight and secured so as to stop the circulation. The indiarubber bandage is then removed, and the limb remains bloodless until the piece of tubing is loosened. The method is excellent if the bandage be applied for only a short time, and if no large cavity be left by the operation, but the application of the bandage leads to paralysis of the smaller blood-vessels, and there is often a troublesome oozing after it has been removed. I have therefore ceased to employ it in such operations as excision of the knee and elbow.

Martin's Bandage is used in the treatment of varicose veins and varicose ulcers, often with excellent results. It consists of a strip of pure black rubber which is absolutely free from sulphur, and is much less stout than Esmarch's bandage, measuring 10½ feet by 3 inches for the leg, and costing about five shillings. The bandage is applied loosely by winding it spirally up the leg as high as may be necessary. It should be put on before the patient gets up in the morning, that is to say, before the veins become overfull. The disadvantages of the bandage are that it is heavy, hot, and waterproof. The perspiration is unable, therefore, to escape, and the bandage often becomes quite wet after it has been worn a short time. A troublesome eczema is thus produced unless the patient is very careful, and she should be advised to wash the limb every night, to dry it carefully, and afterwards to powder the skin with some of the unirritating drying powders used for babies.

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Baths. *See* BALNEOLOGY.

Bed-Sores.—A bed-sore is an inflammatory condition of the skin and underlying structures, produced by a combination of causes, and resulting in local death of the tissues affected. A form of sore, almost identical with the bed-sore, often results from the careless management of various forms of splint (*Splint pressure-sore*).

CAUSES.—Bed-sores are liable to appear in patients who are much devitalised from any such cause as old age, attacks of acute fever, especially typhoid fever, kidney or liver affections, cardiac disease, chronic phthisis or other pulmonary condition, resulting in venous stasis. Any interference with the nerve supply of parts, such as occurs in injuries or diseases of the peripheral nerves, spinal cord or brain, and in some forms of insanity, is a most potent factor favouring the formation of bed-sores.

If to these predisposing causes be added prolonged and excessive pressure, especially on a portion of skin lying immediately over a bony prominence, for example, the sacrum, the great trochanter, the malleoli, the tips of the heels, the shoulder-blades, elbows, or anterior superior iliac spines, the development of a bed-sore is almost assured.

Moisture from the accumulation of skin secretions, the escape of urine, fæces, or wound discharges, promotes the process by improving the conditions favourable to bacterial life, the growth of micro-organisms in these secretions being perhaps one of the most important elements determining bed-sore formation.

Excoriations, which may develop into true bed-sores, sometimes form where two skin surfaces remain constantly apposed, as in the region of the scrotum or labium, under pendulous mammae, or between fingers or toes confined in a splint.

CLINICAL FEATURES.—Two varieties of bed-sore are met with clinically: (*a*) the acute bed-sore; (*b*) the chronic bed-sore.

The Acute Bed-Sore.—This form, which is due to a lesion of the trophic nerves of the skin (Charcot) or to vaso-motor paralysis, comes on very rapidly, sometimes in a few days or even in a few hours, after serious injuries to the spinal cord or brain. It has also been met with in cases of cerebral hæmorrhage, thrombosis, or embolism; in cerebral softening and tumour, as well as in encephalitis and even in meningitis. Areas of gangrene may also occur from lesions of nerve trunks, such as peripheral neuritis. The part pressed upon becomes red and congested, while the surrounding parts are œdematous and swollen. The redness disappears on pressure with the finger, but the skin does not pit. Blisters soon form, and the skin loses its vitality.

The commonest site in cerebral cases is over the upper and inner part of the buttock, on the side opposite the nerve lesion, *i.e.* on the paralysed side; while in spinal lesions the sore usually forms over the middle of the sacrum. Often these acute bed-sores form on parts where there is no pressure, such as the anterior abdominal wall, dorsum of foot, etc.

In the insane a form of acute bed-sore is not uncommon. It occurs most frequently in general paralytics in an advanced stage, and especially when convulsive attacks are frequent, injuries received during the convulsions being the starting-point. In many cases no undue pressure has been possible. Bullæ and vesicles form on such parts as the inside of the knees, angles of the scapulæ, and especially the heels. The skin becomes very red, rapidly loses its vitality, and a black, dry eschar forms and slowly separates, leaving a deep cavity which is very sluggish in healing.

The Chronic Bed-Sore.—The first evidence of an ordinary bed-sore is a dusky, reddish-purple colour of the skin, gradually growing darker, till it becomes almost black. The surrounding parts are œdematous, and vesicles may form, which, on bursting, reveal the papillæ of a greenish hue. A tough grayish-black slough then forms, often painlessly, over the most prominent point of pressure, and this gradually spreads both in width and depth till it may reach the periosteum or bone. In not a few cases the sloughing over the sacrum has extended till it has involved the spinal canal and given rise to a fatal septic spinal meningitis.

After the sloughs separate, a raw unhealthy granulating surface is left, which, however, under appropriate treatment, tends to heal rapidly.

Bed-sores are often the starting-point of general septic infection of a pyæmic kind, which may determine the fatal issue in chronic diseases affecting debilitated persons.

PREVENTION OF BED-SORES.—It would be too much to say that bed-sores are entirely preventible by careful nursing, but there can be no doubt that since the advent of the modern trained nurse, they have become, not only much less common, but also much less severe, than in the days when the care of the sick was largely in the hands of untrained and ignorant persons. Without applying it too rigorously, however, it may be accepted as a safe working rule, that, save in the case of completely paralytic and insane patients, the occurrence of bed-sores must be laid to the account of the nurse.

For the occurrence of splint pressure-sores there is no excuse. The surgeon is responsible for the application of the splint, and in the adjustment of pressure due allowance should be made for any nervous or other debility.

The first essential to the prevention of bed-sores is the regular changing of the patient's position, so that no one part of the body will be pressed upon for any length of time at a stretch. No matter what the primary disease may be, the nurse should vary the position from time to time, unless she receive distinct orders that the patient is *not* to be moved. The changes need not be great—a slight rolling on to one or other side for a short time now and again; a small cushion under the hips at one time, and under the shoulders or small of the back at another; alternately bending and straightening the limbs. Such variations in most cases will be sufficient.

In patients more than usually liable to develop bed-sores, special means must be taken to remove pressure entirely from the bony points which are but thinly covered with soft tissues, and to distribute it widely on the surrounding parts. Large pads of wool, with a hole cut in the centre to receive the prominence; ring-shaped air or water cushions (not too tightly distended) fulfil these purposes if they are regularly attended to and readjusted. In some cases a complete water bed is necessary in addition to these more purely local measures.

By placing the patient on a prone couch, pressure is removed from the parts most likely to become the seat of bed-sores. It is necessary, however, to keep a sharp look-out on the anterior superior iliac spines, knees, ankles, and ribs, lest sores develop in these situations.

Absolute dryness of the skin is another important factor in preventive treatment. Once every day, if not oftener, the susceptible parts are sponged with soap and water, *thoroughly dried*, and then rubbed with methylated spirit, which is allowed to dry on the skin. After this, the part is dusted with powdered boracic acid, this being not only a good drying powder, but also antiseptic, checking the development of bacteria in the decomposing skin secretions. Various emollient applications, *e.g.*, balsam of Peru, resin ointment, boracic ointment, etc., have been suggested as substitutes for dusting powders, but they are on the whole less cleanly and less satisfactory.

In patients who perspire freely, these precautionary measures must be taken more frequently; and when there is incontinence of urine or fæces, as in paralytics, special means must be adopted to prevent wetting of the patient's linen and bed-clothes. Sheets must be changed whenever they become damp, and when a water-bed is in use, a layer of blanket should intervene between it and the sheet to absorb the moisture which is apt to condense on the surface of the indiarubber.

Bandages, wearing apparel, and sheets must be kept free from wrinkles, and crumbs and other extraneous matters must be carefully excluded.

TREATMENT OF BED-SORES.—Once a bed-sore has formed, every effort must be made to prevent its spread. The precautionary measures above described are maintained and if possible increased. The broken surface is kept clean by frequent washing with mild, yet efficient antiseptics, such as boracic lotion, carbolic acid (1 in 40), or lysol (1 in 100). Dry absorbent dressings are applied and frequently changed. It is sometimes found necessary to employ moist or oily substances, *e.g.* boracic poultices, eucalyptus ointment, or balsam of Peru, to facilitate the separation of sloughs, and to promote the growth of granulations.

It has been suggested that the slough should be incised and packed underneath with iodoform gauze. Other surgeons prefer, in moderately strong patients, to excise the slough, scrape the remaining sore, and then sterilise it with pure carbolic acid.

Skin grafting is often useful in covering in the large raw surfaces left after the separation or removal of sloughs.

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Bell's Paralysis. *See* FACIAL NERVE.

Beriberi.

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Definition.—A specific form of peripheral neuritis occurring endemically and, occasionally, as an epidemic of wide distribution. In addition to the usual phenomena of multiple peripheral neuritis, it is specially characterised by localised or general œdema and marked liability to dilatation of the heart.

Nomenclature.—Till recently much confusion existed with regard to this disease, principally on account of its being confounded with other morbid conditions, the diversity of its clinical features, and the multiplicity of names bestowed on it. Now we know that the "beriberi" of India, the "barbiers" of Mauritius, the "morbus innominatus" of Brazil, the "sugar-works sickness" of Bahia, the "bad sickness" of Ceylon, and the "kakke" of Japan, are only different names for the same disease.

The derivation of the term beriberi is uncertain. It was first used in European literature by Bontius, in 1629, and is possibly derived from a Cinghalese word signifying "extreme weakness."

Geographical Distribution.—Our knowledge of this is very incomplete. Formerly believed to be limited to south-east Asia, beriberi is now known to occur here and there practically all over the tropical and a large part of the sub-tropical world. It has also been recognised in higher latitudes, as in Saghalien, in Newfoundland, in the United States, in Australia, and in Ireland. A better knowledge of the disease may have contributed to its recognition in hitherto unsuspected places, but it is also quite possible that its wide distribution in recent times may be owing to the increased communication going on all over the world, and the consequent importation of the infection into places previously immune. We know from Chinese and Japanese records that in ancient times beriberi was a common disease in parts of China, and a rare one in Japan. At present this distribution appears to be reversed; the disease is comparatively uncommon in China and very common in Japan. In the latter country its frequency has enormously increased during the last century. Lately it has appeared, apparently for the first time, in Australia, both among the aborigines and among the immigrant Chinese. It has also broken out in Fiji (in Japanese coolies), in New Guinea, in the Sandwich Islands, in New Caledonia, and in certain other places where it was formerly, if we can trust the records, unknown. The most important centres of the disease at the present day are probably Japan, the Malay Archipelago, and Brazil. In certain of the Netherlands Indies (Java, Sumatra) the mortality from beriberi among coolies, soldiers, and sailors, at certain times and in certain places, is very great. In India and Ceylon in modern times it has become, apparently, a comparatively rare disease. It is quite possible that a proportion of the cases of multiple peripheral neuritis met with in Europe, and which have been attributed to alcohol, arsenic, cold, and the toxines of better-known infectious diseases, are really beriberi. It has been recognised quite recently in certain lunatic asylums in Great Britain, the United States of America, and, possibly, in France.

Epidemiology.—Although confined as a rule to certain limited areas, at times it may extend as a widespread epidemic over vast regions; this was the case in Japan, and in the Brazils in the early sixties. Usually it picks out particular dwellings or particular districts. It is prone in the endemic area to break out among coolie gangs engaged on extensive engineering works; especially where the hygienic conditions are defective, as at the Panama Canal and Congo Railway works. It is very common among the labourers in mines and plantations in the Malay country. The Dutch native troops at Atcheen have also suffered much from this disease. Collective dwellings, such as asylums, barracks, jails, hospitals, schools are prone to become beriberi centres. Ships are specially liable to become infected, the disease often appearing in the crews even on the high seas. Both ships and buildings may retain their infectiveness for years, and that notwithstanding energetic attempts at disinfection. In such buildings or ships, when the warm season comes round, fresh cases of the disease are apt to occur, or old-standing ones to relapse.

The question of the contagiousness of beriberi has been much discussed; so far the data are insufficient to justify a definite conclusion on this point. Apparently the disease may be conveyed from place to place, but in what way it passes from man to man, or in what way the disease is acquired in the endemic area is still unknown. Heat and moisture have a distinct influence on the activity of the *materies morbi;* for whereas within the tropics, in the zone of perennial high temperature, beriberi occurs all the year round, in higher latitudes it occurs only during the summer months, disappearing during the cold season. In the tropics it is most prevalent during the rains. In either tropics or sub-tropics a sudden fall of temperature, especially if accompanied with a damp condition of the atmosphere, is apt during an epidemic of beriberi to be followed by the appearance of fresh cases, and to aggravate the symptoms in existing cases.

Beriberi affects low-lying damp localities, whether it be in the country or in cities.

Predisposing Causes.—New comers to a beriberic district are very prone to the disease; the natives, however, are by no means immune. It attacks the robust as well as the feeble, the well-nourished rich man as well as the half-starved beggar. All ages, both sexes, and all races are liable; the very young and the very old enjoy a relative immunity. Attacks of acute disease predispose to beriberi, so that this disease is often found as a sequel or accompaniment of typhoid, malaria, dysentery, phthisis, syphilis. Pregnant and puerperal women are especially prone to the disease.

Cause.—Nothing definite is known as to the actual agent which produces beriberi. Four sets of hypotheses have been advanced:—(*a*) That it is a form of malaria, of scorbutus, of rheumatism, of anæmia, or that it is a helminthiasis. The facts do not harmonise with any such etiology. These and similar explanations of the phenomena of beriberi have now been definitely abandoned. (*b*) That it is the expression of a type of physiological starvation—nitrogen starvation. The experience of the Japanese navy is quoted in favour of this hypothesis. Formerly the Japanese fleets were annually ravaged by beriberi. On adopting a more liberal dietary the disease almost immediately disappeared from the crews. Lately a Dutch writer attributes the liability of the prisoners in certain jails in Java to the use of decorticated rice—a rice, he maintains, deprived of an important nutritive element. Defective dietary may predispose to beriberi. But that it can cause beriberi is certainly not the case; for, could it do so, the disease would be a feature in every famine, it would affect the poor everywhere, and it would always spare the rich. (*c*) That it is caused by the invasion of the body by a microbe. Many such parasites have been proclaimed, both bacteria and protozoa; but hitherto the evidence for any one of them being anything more than an epiphenomenon or, possibly, the outcome of defective observation, is inconclusive. (*d*) That it is caused by a toxine the product of a saprophyte living outside the human body, certain conditions of temperature, moisture, and of an organic culture medium being necessary for its proliferation. Such a hypothesis seems compatible with most of the peculiarities, both clinical and epidemiological, of beriberi. The analogy to the yeast plant, alcohol, and alcoholic neuritis is very close and suggestive. Such a hypothesis explains the rapid recovery in many cases of beriberi on the patient's removal from a tainted house, ship or locality; a rapidity of recovery hardly compatible with a parasitic theory of the disease.

Incubation.—The incubative period of beriberi has not been definitely ascertained. Recent observations show that the disease may appear in individuals coming from an uninfected locality in four or five weeks after arrival in an endemic centre. Formerly a residence of several months was considered necessary, but the observations referred to tend to show that in highly infective localities a much shorter time suffices for the development of symptoms.

Symptoms.—These vary very much according to the type of the disease, the stage of the disease, the intensity of the infection, and the occurrence or otherwise of intercurrent reinfection.

Briefly, the clinical picture is one of a multiple peripheral neuritis, most marked in the lower extremities but very frequently extending to the hands and arms. To the paretic, paræsthesic, and analgesic symptoms usual in this condition a greater or less amount of œdema, varying from slight puffiness over the shins to general dropsy, is superadded. Besides this, the almost invariable occurrence of breathlessness and palpitation affords evidence of the frequency in beriberi of implication of the neuromuscular elements of the circulation, and especially of the heart.

Beriberi may commence suddenly; the patient, for example, having gone to bed apparently quite well, may wake up in the morning with the symptoms well marked. More generally there is a period of days or weeks during which vague pains in the limbs are experienced, together with muscular debility coming and going, a degree of palpitation, perhaps diarrhœa, and it may be a slight but evanescent rise of temperature. Rapidly, or more slowly, the feebleness of the legs and, generally, a very evident pre-tibial puffiness increase. The muscular weakness, always most marked in the muscles of the legs, may now rapidly or more slowly extend to those of the thighs, of the hands and arms, and, in a proportion of instances, to those of the trunk, including the diaphragm and intercostal muscles. The affected muscles are tender on pressure—sometimes exquisitely so, and exhibit the reaction of degeneration on being tested electrically. The knee-jerks, at first exaggerated, disappear in three to six days. The paresis may be slight or it may be complete, and may advance or recede slightly from time to time. According to its degree, the patient will exhibit merely weakness of the limbs, or he may be completely bed-ridden and quite unable to move legs or arms.

Similarly the œdema, which is present at the outset in practically all cases, may amount merely to a local puffiness over the shins or it may rapidly pass to general dropsy, the patient in the latter instance presenting the appearance of one suffering from acute nephritis. Usually the œdema amounts only to considerable facial swelling, distinct œdema of feet and legs, œdema about the root of the neck and over the sternum, and, almost always, œdema over the sacrum.

At the same time various paræsthesiæ—tingling, burning formication—are experienced in the skin over the paretic muscles, more especially over the front of the legs and on the dorsa of the feet, the front of the thighs, the finger-tips, and around the mouth. In these localities common sensation is defective, sometimes completely in abeyance. The muscles, particularly those of the calf and some of the nerve trunks, are found to be exquisitely tender on pressure, and in certain cases the implicated muscles are from time to time attacked with painful cramps.

Concurrently with the development of the paresis, and more especially in such cases as show well-marked dropsy, the patient becomes liable to palpitations, attacks of alarming breathlessness, and of precordial distress—particularly after a full meal, or on exertion.

In a proportion of instances physical examination may or may not discover evidences of effusion into the pericardium or pleuræ, more rarely into the peritoneum. The heart's impulse is diffuse and the precordial area may be distinctly enlarged, especially towards the right. The action of the heart may be irregular; it is always remarkably quickened by movement or the erect posture. Palpitations, throbbing of the vessels in the neck, and epigastric pulsation may be very marked, whilst the pulse at the wrist may remain feeble. Bruits, generally systolic in rhythm, are usual; they come and go, changing, it may be, from hour to hour. The spacing of the intervals is peculiar, the first and second intervals being in many cases practically equal. The first and second sounds are often hard to distinguish, and one or both of them may be reduplicated. Generally there are well-marked signs of dilatation of the right ventricle and tricuspid regurgitation. Œdema of the lungs, effusion into pleuræ or pericardium, may occur and place life in danger. Or it may be that paresis of the intercostal muscles, or paresis of the diaphragm, supervenes. Sometimes the dilatation of the heart rapidly increases. In either event orthopnœa sets in, and, if the physical condition be not quickly relieved, death may take place within a few minutes of the appearance of the urgent symptoms.

The course of beriberi is very uncertain, particularly so if the patient be not removed from the endemic centre. If placed in healthy surroundings, provided nerve and muscle degeneration have not proceeded too far, usually, after a few days, improvement begins. In all cases in which the muscular atrophy is considerable many months must necessarily elapse before the patient regains full use of his limbs. In a few instances permanent deformity, such as talipes equinus, ensues, or some degree of dilatation of the heart may persist.

In the dropsical form—often called "wet" beriberi—during the active phase of the disease the urine is generally very scanty, amounting to only a few ounces in the day; though highly coloured and of high specific gravity, it rarely contains albumen. Attacks of dyspnœa, in which the patient has to sit up in bed and struggle for breath, occur from time to time. During these the extremities may become cold, the lips blue and the face livid; and whilst the heart can be seen to be acting violently, and the vessels of the neck pulsating strongly, the radial pulse is but small and feeble. Death frequently takes place during one of these attacks, the fatal issue manifestly depending on paresis and dilatation of the right heart. Pain under the sternum of a most distressing character generally accompanies the orthopnœa which may supervene without obvious cause, but which is readily provoked by muscular effort, or by the pressure on the heart of the stomach after a full meal. When a "wet" case is about to improve active diuresis sets in; the dropsy may disappear entirely in forty-eight hours, the patient's shrivelling limbs now showing, in many instances, a well-marked muscular atrophy.

In those cases in which paresis is the principal symptom—"dry" beriberi as it is sometimes called—the progress of events is equally uncertain so long as the patient remains in the endemic area. The symptoms may be of the most trifling description, amounting to slight weakness and numbness of the legs, or legs and arms, tenderness of the affected muscles, and slight muscular atrophy. On the other hand the paresis may be complete and the atrophy extreme, involving nearly every voluntary muscle in the body except those of the eyes, face, mouth, and gullet, which are very rarely attacked. The panniculus adiposus in such cases is also atrophied; so that in extreme instances the patient, reduced to skin and bone, lies helpless in bed unable to move hands or feet, and quite unable to feed himself. He can speak, however, masticate and swallow, urinate and defæcate, is free from fever, and may, apart from the paresis and muscular tenderness, feel perfectly well. He may lose his voice from implication of the laryngeal muscles; his breathing may also be affected from paresis of the diaphragm or of the intercostal muscles. Effusion into the serous cavities is comparatively rare in this type of beriberi; urgent cardiac symptoms, although common enough, are also less frequent than in "wet" beriberi. The progress of such cases may be slow or rapid. In consequence of the high degree of atrophy complete recovery is necessarily a matter of months, and, if the patient remain in the endemic area, may be frequently interrupted by repeated relapse.

When a case shows well-marked œdema and, at the same time, well-marked muscular paresis, it is said to be one of "mixed" beriberi. In such a case, on the disappearance of the œdema, the atrophy and hyperæsthesia of the muscles are found to be marked.

One type of beriberi may suddenly or more slowly assume the clinical characters of another—a "wet" case become "dry," or, *vice versa*, a "dry" case become "wet." Of the three types the "wet" form is the more dangerous, the "dry" being, as a rule, the less dangerous. Death, however, may occur, usually with surprising suddenness, in any of the types. It may be brought about by rapid increase of the dilatation of the right side of the heart—usually more or less marked in all cases of active beriberi; by syncope; by œdema of the lungs; by effusion into the pericardium or pleuræ; by paralysis of the muscles of respiration; or by a combination of these conditions. Implication of the pneumogastric, phrenic, or cardiac nerves—the pathological condition on which the fatal issue of these cases depends—takes place quite suddenly. It may occur in what appears to be in other respects a mild case; consequently prognosis in beriberi should always be very guarded.

Mortality.—The mortality varies much according to the epidemic and the treatment. In some epidemics it has amounted to 50 per cent of those attacked; in others it has not exceeded 5 per cent. It is always highest where the patients are allowed to remain after attack in the endemic centre; and especially so when the patients are crowded together in the building or ship in which their disease was contracted, and in which, it is to be presumed, poisoning is still going on.

Morbid Anatomy.—The post-mortem appearances vary with the type of case. In dropsical beriberi there is, of course, a large amount of serous infiltration of the tissues, and very often considerable serous accumulation in the pleuræ, pericardium and, more rarely, in the peritoneum. Œdema of the lungs is not unusual. In the paretic, atrophic, "dry" type, on the other hand, the tissues are abnormally dry. In both types the heart, more especially the right side, is dilated and, perhaps, slightly hypertrophied. The right cavities and associated blood vessels are distended with blood and may contain ante-mortem clot. Ecchymoses in the serous and mucous surfaces and, occasionally, in the muscles and nerves, are common.

Baelz and Scheube were the first to demonstrate the neuritis which is the fundamental lesion of this disease. The results of these observers were subsequently confirmed by Pekelharing and Winkler and others. The neuritis specially affects the motor fibres, and is always most marked at the periphery, diminishing in intensity as the nerve is traced towards the cord into which, according to most observers, the process does not extend. The microscopic characters of the lesion are those usual to multiple peripheral neuritis, and need not be described at this place. The muscles supplied by the affected nerves will be found to have undergone various degrees of degeneration. In some spots there are evidences of myositis; such have been specially observed in the cardiac muscle.

Diagnosis.—In the tropics and sub-tropics, and in ships which have at some time visited the endemic centres of beriberi, epidemics of peripheral neuritis are almost invariably attributable to beriberi. Strachan (*Practitioner*, 1897, p. 477) has described a form of endemic neuritis, very common in certain districts of Jamaica, which, judging from the absence of œdema and the invariable implication of the cranial nerves, is not beriberi; but, with this exception, so far as is known at present, the generalisation just stated holds good. During such epidemics mild degrees of peripheral neuritis, such as are evidenced by slight numbness of the skin of the legs, varying degrees of myalgia and weakness of the limbs, tenderness of the calf muscles, a tendency to palpitations and breathlessness, and slight œdema of the shins, are frequently encountered. Such cases are probably mild or larval forms of beriberi which may or may not proceed to fuller development. In well-marked cases the history, the combination of œdema, palpitations, muscular weakness, tenderness and wasting, the absence of ocular symptoms, and the more rapid evolution of the disease, suffice to distinguish beriberi from locomotor ataxia. The absence of fever, of profuse sweating, the localisation of the pain in the muscles, the absence of knee-jerk, and the presence of well-marked evidences of peripheral neuritis distinguish beriberi from rheumatic fever. The absence of albumen from the urine, besides the presence of the nervous symptoms, distinguish it from nephritis. Although diagnosis is by no means difficult, it is curious to note how frequently, either from carelessness or ignorance, beriberi is overlooked, and very often overlooked with disastrous consequences. It is difficult, however, even with care, in the case of alcoholics exhibiting symptoms of peripheral neuritis while resident in a beriberi locality, to be sure as to the particular poison to which the symptoms are attributable. But even in such a case the presence of marked œdema, of palpitations, and of cardiac bruits would suggest a diagnosis of beriberi; their absence, and the presence of the well-known symptoms of chronic alcoholism, alcoholic neuritis.

Treatment.—The most important measure in the treatment of beriberi is immediate removal of the patient from the spot in which the disease was acquired. As a rule, the carrying out of this measure is followed after four or five days by steady improvement. If the patient cannot be removed he should at least be placed in an airy, well-lighted, and dry apartment well off the ground, if such be available.

The diet should be varied and nutritious but not too bulky. Experience is against the use of rice in this disease; some other cereal should be substituted, both in the case of those already attacked and in those compelled to live in the infected area. If there be no diarrhœa it is well to keep the bowels free by some mild saline aperient; in dropsical cases the amount of fluid taken should be restricted.

Small doses of digitalis or of strophanthus seem to be of service where the heart's action is weak and irregular. Besides these, many other drugs have been recommended in the routine treatment of beriberi; so far the evidence in favour of any one of them is of the slenderest description.

Malarial, dysenteric, syphilitic, and other complications should be dealt with in the ordinary way. If it is found from an examination of the fæces that the alimentary canal is the seat of some form of verminous invasion, this should receive attention. Ankylostomiasis, especially, should not be neglected, particularly if the patient is markedly anæmic.

With a view to preventing or mitigating the cardiac attacks which constitute the principal danger in beriberi while the disease is active, the patient should be kept in bed and warned against over-eating or unnecessary exertion. His attendant should be provided with nitrite of amyl and solution of nitro-glycerine, and be instructed to administer at once one or other of these drugs on the slightest sign of the approach of dyspnœa or other evidence of embarrassed circulation. In these circumstances three to five drops of the one per cent solution of nitro-glycerine may be given every quarter or half hour until dyspnœa is relieved. These measures failing, dyspnœa increasing, and the patient becoming cyanosed, there must be no hesitation in opening a vein and bleeding to the extent of eight or ten ounces. Very often, as a consequence of venesection, relief is immediate though, too frequently, only temporary. The bleeding may be repeated several times if the cardiac symptoms persist. It sometimes saves life.

Pleural or pericardial effusions should be aspirated if they seem to be interfering with the action of the heart.

When acute symptoms have subsided and the muscles are no longer tender, their rehabilitation may be favoured by gentle massage and faradisation. A sea-voyage has often a powerful influence both in arresting the progress of the active disease and in favouring convalescence.

In the event of beriberi breaking out in a school or similar institution the various members of the community should be sent to their respective homes, and the buildings they had inhabited thoroughly disinfected. Ships in which beriberi is known to occur should have their woodwork thoroughly scraped and painted; at the same time, all rotten timbers, foul bilge water, and any organic matter which might be supposed to harbour the germ of the disease should also be removed.

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Biceps Tendon, Dislocation of. *See* SHOULDER-JOINT.

Bile and Biliousness.—The ancients placed bile upon an exalted pedestal. They ascribed to it various properties and powers exercised over the bodily processes. In the Vedas the Hindu poets describe pitta or bile as digesting the food in the stomach, and as able to purge the bowel,—the latter opinion according closely to modern teaching. Galen credited the spleen with the function of secreting black, the liver of forming yellow, bile. Black bile combined the elements of warmth and dryness, yellow bile of cold and dryness; the hot intemperament (our bilious habit) accompanied excess of bile formation, the cold intemperament indicated lack of bile, or the formation of yellow bile alone. Black bile was responsible for melancholia, although, if yellow bile was converted into the black variety, mania resulted. Even as recently as in Cullen's day, the bile was supposed to possess properties derived from the presence in it of humours formed by the liver.

An account of the physiology of the bile-secreting and bile-expelling mechanisms will be found in the article "Liver."

Biliousness.—Popularly biliousness denotes a condition of the body wherein too much bile is secreted by the liver, and too little expelled from the bowel. Medically, the term is only a refuge in time of perplexity, or a help when a vague explanation seems advisable. A "bilious attack," so called, is almost invariably the result of some acute catarrhal condition of the gastric or duodenal mucous membrane, the frequent presence of bile in the vomited matter in such cases arising from reversed peristaltic movements of the duodenal muscular coat, which in turn are induced by the inflamed state of the mucous lining, aided by the irritation and excitement following upon violent emetic efforts. The "bilious" headache, so commonly spoken of, may be due to nervous storms, as in hemicrania, or migraine, auto-intoxication from the absorption of poisonous products from the bowel, or may be a premonition of an impending jaundice from various causes, which may or may not be removed before actual jaundice results, but which does not lie so much in any excess of bile formed by the liver as in some bar to its access to the bowel. When the functions of the hepatic cases are more or less inefficiently performed, less than the normal amount of bile may be excreted, and those substances from which it is manufactured will in part be carried in the general blood-stream. This condition is very different from "biliousness," for there is a lack of bile not an excess, while the symptoms approach very nearly in many cases the accepted signs of the latter, although due chiefly, it is probable, to the presence of poisonous substances in the blood. whose decomposition into innocuous compounds forms no unimportant part of the hepatic duties in health.

A "sluggish liver" generally stamps its possessor as one who eats too much and too rich food, takes too little exercise, and is careless about the evacuation of his bowels. A "bilious temperament," with a sallow skin and indigestion, probably arises from an excess of pigmentary matter in the skin, and a weak digestion, usually overtaxed, with a chronic state of mild auto-intoxication superadded. A "bilious attack" spells gastric or duodenal catarrh, or both. The chronic tinging of the conjunctiva with yellow, so often regarded as a proof of a bilious habit, is only a sign of some difficulty attendant upon the passage of bile in its course from the liver cells to the bowel, which disturbs the pressure balance of its secretion.

In treating "biliousness" the present course of the affection has first to be determined, then dealt with *secundem artem.*

Bile Passages, Affections of. *See* GALL BLADDER, LIVER.

Bilharzia Hæmatobium. *See* PARASITES.

Bites. *See* WOUNDS.

Bitter Almonds. *See* TOXICOLOGY.

Blackwater Fever.

NOMENCLATURE.—Pernicious remittent fever, malignant congestive fever, malignant bilious fever, bilious remittent fever, yellow remittent, yellow chill, hæmorrhagic malarial fever, purpuræmia, hæmatinuria, bilious hematuric fever, hæmoglobinuric fever, bilious hæmoglobinuric fever, epidemic hæmoglobinuria, infantile hæmoglobinuria, paroxysmal hæmoglobinuria, melanuric fever, blackwater fever.

DEFINITION.—An acute infectious disease characterised by an intense destruction of red-blood corpuscles and clinically marked by paroxysms of fever accompanied by rigors, bilious vomiting, jaundice, and hæmoglobinuria.

SYMPTOMS.—We know but little of the incubation period in blackwater fever. The disease may certainly remain latent for a considerable time. Scott, in British Central Africa, noticed that the attack usually occurred about eight days after exposure to certain districts of the low river country.

For a day or two there may be prodromal symptoms, such as general malaise, headache, nausea, and inability to work. As a rule, however, the invasion is abrupt and marked by a severe rigor, which may last from fifteen to thirty minutes or even longer. The temperature usually rises to

103° or 104° F., but sometimes it may attain from the outset 105° or 106° F. The patient is extremely thirsty and much troubled by nausea and persistent bilious vomiting. The vomited matters are yellow at first, but they soon become of a characteristic deep green colour; in grave cases they may assume a dark brown hue from admixture with blood. He usually complains of pain in the lumbar and hypochondriac regions. The spleen and liver are found to be somewhat enlarged and tender to pressure. After two or three hours, the skin and conjunctivæ appear jaundiced, and in most cases the discoloration goes on deepening to an intense lemon-yellow or saffron colour. Meanwhile the urine assumes a dark red colour. It may become very scanty, thick and viscid like molasses, and as black as porter. In bad cases it may be entirely suppressed. There is usually constipation, but sometimes there may be diarrhœa, and the stools may be bilious, or black and tarry.

After a longer or shorter period, usually five or six hours, the temperature may fall with profuse sweating; the patient will feel better and the urine will become lighter in colour and more abundant. But a fresh rigor may supervene and the temperature will rise again with aggravation of all the symptoms. Two or more paroxysms may thus follow in quick succession, or there may be only one paroxysm recurring at about the same time on two or more successive days, or again there may be an intermission of one or more days between each paroxysm. When the paroxysms are severe and frequent, the temperature may not fall lower than 100° F. during the interval, but when they are mild and far between, the temperature falls very soon to normal.

After a few paroxysms, or after the disease has persisted for three, four, five, or six days, the patient may get well spontaneously. But frequently the attack is severe. Several paroxysms may follow in quick succession; the patient becomes very weak and restless; his pulse becomes rapid and out of proportion to the temperature; his breathing becomes laboured and sighing—troublesome hiccough sets in. There may be stupor or intense restlessness, and death may take place on the fifth or sixth day, sometimes as early as the second or third day. When recovery takes place, the temperature falls, the vomiting ceases, and the urine soon returns to normal in quantity and colour. But the discoloration of the skin may not entirely disappear before ten days or a fortnight, and the patient may remain weak and anæmic for a considerable period.

The *death-rate* varies greatly in different places; it is about 60 per cent in British Central Africa (Scott); 50 per cent on the Gold Coast (Reynalds); 22·4 per cent in Greece (Spiridon Kanellis).

Blackwater fever is extremely liable to relapses, which may occur weeks or months after the first attack. They may be very frequent and are easily provoked by over-fatigue, and exposure to cold, so long as the disease is "in the system." Each relapse will be, as a rule, milder than the previous one, but they will all exhibit in a greater or less degree the same symptoms as the first attack. Each relapse may consist of one or more paroxysms. The symptomalogy of blackwater fever is frequently obscured by complicating summer-autumn or tertian infection.

If the *blood* be examined at the beginning of an attack, the red corpuscles are found to be undergoing rapid destruction; their number may fall in less than twenty-four hours to a third of the normal. But the destruction is soon followed by a rapid regeneration; thus blood corpuscles of every size and nucleated corpuscles will be found under the microscope. At the same time will be witnessed a marked leucocytosis, which begins with the paroxysm and persists for a certain length of time thereafter; it is in every way similar to that which meets the invasion of the *spirillum Obermeiri* in relapsing fever. Various hematozoa have been found in the blood of blackwater fever, amongst them *hæmamœba vivax.* But Fisch, Plehn, Woldert, and other observers who have examined the blood repeatedly in blackwater fever cases, seem to have found more frequently a peculiar organism which is very probably the cause of the disease. It is a small unpigmented protozoon possessed of active amœboid movements, and assuming mostly an almond or pear shape. Woldert found about one protozoon in every fifty corpuscles. He noticed that in some cases two parasites having the same shape occurred within the same red corpuscle, and found that the larger sizes sometimes contained one small dotlet of pigment. If freshly-drawn blood be allowed to stand, the serum will be found to present a very dark red colour, indicating the presence of much colouring matter in solution.

The urine in blackwater fever is generally strongly acid. Its specific gravity varies inversely to the amount passed. It is usually above normal. When seen by reflection, it is almost black; when examined by refraction, it is a dark claret colour. On shaking, it produces a reddish-yellow foam. On standing, it deposits a thick reddish-brown sediment, which is chiefly composed of an amorphous yellow substance (destroyed blood corpuscles), aggregated into rounded masses or forming regular casts of the renal tubules. By gradually raising the urine to the boiling temperature a reddish-brown coagulum is formed, which consists mostly of cell-globulin. Occasionally a few corpuscles of normal appearance may be detected in the sediment, but as a rule they are totally absent. Of course nephritis may be exceptionally associated with blackwater fever as with tertian fever, and then hæmaturia may complicate or follow hæmoglobinuria, and the urine will contain normally-shaped blood corpuscles and serum albumen. The spectroscopical examination exhibits the two absorption bands of oxyhæmoglobin.

Yersin found a bacterium in the urine of his patients, and claimed it to be the cause of blackwater fever, but Breaudat recognised it to be *bacillus coli communis.* Lion noticed the presence of *proteus vulgaris*, which is a common putrefactive organism.

MORBID ANATOMY.—The seat of the disease is the *blood*, in which large numbers of red corpuscles are attacked and rapidly destroyed. This destruction seems to occur chiefly within the capillaries of the internal organs. It is similar to that which follows on the transfusion of blood from an animal of a different species. There is always a marked polynuclear leucocytosis.

The liver, spleen, and kidneys are enlarged, and their congestion is on account of the increased functionality demanded for the reduction and elimination of the infected blood corpuscles.

The *liver* is of a yellowish-brown colour on section; the cells of its parenchyma are enlarged, granular, proliferating (cloudy swelling). Many contain particles of a yellowish substance (reduced blood corpuscles). The gall bladder always contains much bile and may be greatly distended.

The *spleen* is congested; its capsule is thick and the parenchyma is black and soft. Masses of dark yellow substance are to be found both between and within its cells.

The *kidneys* show intense hyperæmia. The cortical substance on section is of a pale yellowish colour, the pyramids showing brown streaks, especially towards their apices. There is marked infiltration of leucocytes in the interstitial tissue, and the epithelium of the secreting tubules shows cloudy swelling. The convoluted tubules are filled with granular pigment substance which is being excreted by the lining cells. This yellowish substance, together with shed epithelium, often forms regular casts within the straight tubules. The suppression of urine which takes place in bad cases is obviously due to the blocking up of the tubules.

GEOGRAPHICAL DISTRIBUTION. — Blackwater fever has a very wide distribution, but its prevalence in various countries is most unequal. It is prevalent throughout tropical Africa. It is found all along the West Coast from the Senegal to the Coanza, but principally on the Congo, and on the deltas of the Niger and Gambia rivers. On the East Coast it is also widely spread, especially along the Zambesi, the lower Shiré, and the shores of Lake Nyassa. It is common in some parts of Madagascar. In America it extends over the Southern States of the Union, chiefly Florida, Georgia, Alabama, Mississippi, Arkansas, and Texas, but recently also in North Carolina and Virginia. It is also prevalent in the plains of Venezuela, and in the West Indies. In Europe its principal seats are the Greek Archipelago, Sicily, and Sardinia. In Asia the French have found it in Tonquin, and lately it has been described as occurring in Assam, Darjeeling, Terai, Meerut, and Amritsar. In regard to India it is interesting to note that the disease had not been described previous to 1885, and this would lead us to believe that it might be of recent importation. The disease exhibits such striking symptoms that it would be absurd to think that it could have escaped the attention of such men as Chevers, Carter, Martin, Fayrer, Morehead, and Maclean, had they met with it. Powell, who informs us of its appearance in Assam, states that he saw the disease previously "in those who returned from the Gold Coast." American authors state that it has lately become more prevalent in the United States. Meek says that it first appeared in Texas in 1866. It would be interesting to know whether it existed in America previous to the advent of the African negro.

Although blackwater fever has a wide distribution it is limited in its endemicity to low swampy grounds. Some authors have reported its occurrence at high altitudes, but that does not prove that it was contracted on the highlands. We know that the disease may remain latent for a long while, and that those who have suffered from it may have relapses at long intervals and far from the place in which the disease was contracted. It is not to be wondered at, therefore, that the disease should develop sooner or later in those who were obliged to cross the swamps at the foot of mountains in the season in which the disease is rife. However, I do not deny that the disease might be contracted exceptionally in the highlands, because if the intermediate host of the blackwater fever organism were a flying insect, it might be blown by the wind on to the highlands, and if it were a parasitic insect it might be introduced with imported cattle.

SEASONAL PREVALENCE.—In the Southern States of the American Union, blackwater fever is reported to be especially frequent in late summer and in autumn. On the West Coast of Africa it prevails after the rainy season is over (August and September), when the atmosphere is hot and moist. It is therefore apparent that, like other infectious diseases, blackwater fever is directly or indirectly dependent on peculiar meteorological conditions. Of course relapses may occur at any season and in any climate.

EPIDEMIOLOGY.—Blackwater fever at times assumes an epidemic form. It may not be seen for years in a district, and then numbers of cases may occur within a short time. It has been observed that the disease has of late years become more prevalent in some regions, while in others it seems to have been only recently introduced. It broke out among the labourers employed in making the canal through the Isthmus of Corinth. Dr. Wenyon, of Fatshan, China, says that in 1885 "it ravaged like a plague the Chinese army on the Tonquin border of Krvangsi." In collective dwellings, such as barracks, hospitals, schools, it may attack several persons at the same time. In 1885 it broke out in a prison in Castiades (Sardinia).

PREDISPOSING CAUSES.—Sex and age are of no importance; adult men are chiefly attacked on account of their greater exposure. At one time race was considered an important factor. In Africa, Europeans and Chinese are attacked in great numbers, while the natives are said to enjoy an immunity. That the natives should be relatively immune is quite natural, but it is not a racial immunity; it is simply the immunity that a long struggle through many generations has conferred on those who inhabit the districts in which the disease is endemic. In fact, natives of the same tribe, but living in places which are usually free from blackwater fever, will contract the disease just like Europeans when they come within its reach. Plehn mentions a serious outbreak of blackwater fever among the blacks in the Cameroons, the disease specially attacking those who had come to the coast from the interior. Reynalds informs us that the disease occurs in severe epidemics amongst the natives of Ashanti.

The chief predisposing cause to blackwater fever is debility from previous illness. It is usually those who have suffered from summer-autumn infection, tertian fever or dysentery, that become the victims of blackwater fever. Plehn, Scott, Ritchie, Lynch, and many other observers have mentioned cases of the disease without antecedent malaria in robust individuals of recent arrival, but that previous debility predisposes to blackwater fever, as to beriberi and other diseases, is a well-established fact.

It has been repeated by all those who have written on blackwater fever that Europeans are rarely attacked within the first year of residence on the African Coast, and this fact has been emphasised by those who wanted to prove the theory that hæmoglobinuria was simply a feature of ordinary malaria in cachectics. We know now of cases which occurred very shortly after landing, but that the disease should usually attack new-comers several months after their landing is what might be expected, because it can only be contracted in the season and districts in which it is prevalent.

ETIOLOGY.—At one time blackwater fever was considered one of the manifold manifestations of so-called "malaria." This opinion is not yet universally abandoned, but the writer believes that recent progress achieved in the study of tropical diseases conclusively indicates that this view is not to be entertained.

A popular opinion in Sicily and Sardinia is that the disease is caused by the eating of fresh broad beans, because it is at the time of the ripening of *faba vulgaris* that the disease is rife.

Professor Tomaselli of Catania being aware that the clinical features of blackwater fever differed widely from those of the co-endemic malarial diseases, and having noticed that the administration of quinine frequently aggravated the blackwater paroxysms, or seemed to provoke new relapses, argued that the disease was not malaria *per se*, but a form of quinine poisoning peculiar to malarial cachectics.

In 1896, Bastianelli, while endorsing Tomaselli's old theory, stated that there was a hæmoglobinuria which could not be attributed to quinine, and which he believed to be a peculiar manifestation of intense æstivo-autumnal infection.

Tomaselli's theory was almost discarded, when Koch suddenly reinstated it by asserting, in the most emphatic manner, that blackwater fever was "nothing more nor less than quinine poisoning."

As early as 1893 Dr. Patrick Manson, at a meeting of the Epidemiological Society, stated that he believed blackwater fever to be a disease *sui generis.*

At the last Edinburgh meeting of the British Medical Association, and in a paper contributed to the Journal of Tropical Medicine, the writer endeavoured to prove that blackwater fever is a specific disease, clinically and probably also etiologically akin to *Texas fever* or *redwater fever* of cattle, and he likewise observed that *paroxysmal hæmoglobinuria* and *infantile* or *epidemic hæmoglobinuria* are possibly other names for the same disease.

Theories.—The theory that blackwater fever is due to some toxic substance contained in broad beans need hardly be discussed. *Faba vulgaris* is eaten the world over, and it would therefore be difficult to understand how it should give rise to disease only in some few places, and in such places only in a very small minority of those who feed upon it. Then again, this theory does not account for the great prevalence of the disease in places in which this plant is not cultivated. It is, however, important to note that this popular belief arose in countries greatly infected by malarial diseases, and shows, therefore, that the natives consider blackwater fever a distinct pathological entity.

The theory of cold, in this as in many other diseases, arose from the chilly feelings which are experienced by the patient in the period of incubation, and the severe rigor which ushers in the attack. But these chills and rigors have nothing to do with the external temperature; like those of pneumonia, relapsing fever, tertian fever, and pyæmia, they are connected with the active phases of their special parasites. With the advance of bacteriology, cold has lost all importance as an etiological factor.

The idea that quinine is the cause of blackwater fever arose in Sicily, where the disease is not very frequent. The English physicians who have had much experience with the disease in intertropical Africa, are unanimous in protesting against it. Quinine never produces hæmoglobinuria, not even when administered in highly toxic doses.

Tomaselli's theory of blackwater fever was founded on the fact that the administration of quinine aggravated the hæmoglobinuric paroxysms and provoked new relapses. Studying very carefully the literature of so-called "quinine hæmoglobinuria," we find the following to be the real facts.

In regions in which blackwater fever is endemic, a patient may have suffered repeated attacks of tertian fever, or of summer-autumn fever, which were satisfactorily treated with quinine. One day, suddenly, instead of an ordinary malarial paroxysm, blackwater fever manifests itself, and the continued administration of quinine seems, in some cases, to aggravate the disease, in others it appears to have a beneficial influence.

The first attack of blackwater fever being well over, the administration of quinine may or may not be followed by a relapse, or the relapse may occur without the administration of quinine.

Later, the hæmoglobinuric infection, being seemingly spent, the administration of quinine, even in large doses, never causes it again, as it previously seemed to do.

Thus we see that the connection of quinine to blackwater fever is not one of cause and effect, but merely one of coincidence. But the fact which cuts this theory at its very root is that blackwater fever breaks out at times in veritable epidemics amongst the natives of intertropical Africa, who are recognised to enjoy a certain immunity against malaria, and who certainly do not take quinine.

The oldest and more widely accepted theory is that which considers blackwater fever to be the manifestation of a specially intense attack of "malaria."

The admirable studies of the Italian school (Golgi, Marchiafava, Celli, Bignami, and others) which isolated tertian fever and quartan fever from the intricate group of so-called "malaria" made it evident years ago that blackwater fever had nothing to do with these two diseases, and it was argued, therefore, that it must be a manifestation of severe æstivo-autumnal infection. But now we know that several diseases are confounded under the most inappropriate name of summer-autumn fever. In Italy Grassi and Feletti distinguish three separate parasites in this group. Careful microscopical studies in other regions will probably lead to the discovery of other varieties, the existence of which may already be surmised from the different clinical features which the fevers of various places assume. We are therefore led to the conclusion that hæmoglobinuria cannot be the manifestation of a more intense attack of all and any of the already differentiated diseases, but that it is the peculiar and constant characteristic of one specific and as yet undetermined parasite.

Recent studies in "malarial" and other diseases have taught us that each species of parasites gives rise to peculiar symptoms which are always the same in kind, and differ only in degree. The difference in symptoms between tertian, quartan, and the ordinary malignant tertian of most tropical countries (æstivo-autumnal infection) is small when compared with that which exists between any of these diseases and blackwater fever.

To expect a different symptomatology in a more severe attack of summer-autumn fever is not in accordance with fact. In the most pernicious cases of summer-autumn fever we never find the symptoms peculiar to blackwater fever, and, on the other hand, all cases of blackwater fever, however grave, however mild, always exhibit the very same symptoms, with no other difference than that of intensity and duration.

Blackwater fever is extremely liable to relapses. Each relapse will be usually milder than the previous one, but they will all exhibit the very same symptoms of the original attack, thus proving, in the most evident manner, that to believe a mild relapse of blackwater fever to be a more severe attack of æstivo-autumnal infection is an untenable paradox.

The reasons that lead to the belief that blackwater fever was a manifestation of a more intense infection of "malaria," are—

1. The disease is found in malarious regions.
2. It attacks those who have previously suffered from malarial diseases.
3. The parasites of malaria have been found in the blood of blackwater fever patients.

We have already stated that, though co-endemic in several places, blackwater fever and the malarial diseases have their peculiar geographical distribution.

That those who have suffered from ordinary forms of malaria are chiefly attacked by blackwater fever is certainly no proof of the identity of the two diseases. We do not believe tuberculosis to be a peculiar manifestation of typhoid fever simply because it frequently follows in its wake. Experience has taught us unmistakably that if at the fall of the rainy season we send to the Congo or to the lower Shiré or to Lake Nyassa a man who has suffered from malaria in India, China, or Australia, he will contract blackwater fever almost to a certainty, but that he will run no such risk however long he remain in the malarious districts of Asia and Australia.

The fact that malarial parasites have been found in the blood of patients suffering from blackwater fever is no better proof of identity. That we should find the tertian, quartan, and "æstivo-autumnal" parasites in blackwater fever is what might be expected, for the ordinary malarial diseases and blackwater fever are in many places co-endemic, and, all being debilitating diseases, one predisposes to the active manifestation of a latent infection of the others. No one would believe *anchylostoma duodenale, tænia saginata,* or *filaria nocturna,* to be the cause of blackwater fever simply because they had been found in a blackwater fever patient. The very fact that both *hæmamæba vivax* and the parasites of the summer-autumn group have been found, greatly invalidates the theory of regarding any of them as the causative agents of hæmoglobinuria.

Several observers noticed that the parasites which they most frequently found in cases of blackwater fever differed somewhat from those described by authors in malarial diseases, and they suggested that the parasites of hæmoglobinuric fever might be a variety of the ordinary æstivo-autumnal parasites.

It seems to me that their descriptions of the parasites found in blackwater fever correspond exactly to those of the hematozoa found by Smith and Kilborne in the hæmoglobinuric fever of cattle (Texas fever).

The observations of the two American authors were confirmed by Krogius and O. von Hellens in Finland, Sanfelice and Loi in Sardinia, Celli and Dionisi in the Roman Campagna, and Koch in East Africa.

The organisms found in blackwater fever and Texas fever are both small unpigmented protozoa, both show active amœboid movements, both assume usually the shape of an almond or pear, both are frequently found in pairs in the same corpuscle, both affect the red corpuscle in the same way and give rise to identical symptoms, both are found in the same season and in the same swampy districts.

Texas fever, or better, *redwater fever*, is found not only in oxen, but in horses, sheep and possibly in other animals. It is not improbable, therefore, that it should also attack man as is the case in anthrax, tuberculosis, and other diseases which have a wide zoological distribution.

The hæmoglobinuric fever of cattle is conveyed by a tick (*Boophilus bovis*) which frequently infests other mammals (sheep, goats, horses, mules) and man. The writer has been frequently attacked by ticks while botanising in Southern Italy.

In the United States, in Sardinia, in East Africa, and in other regions we know that hæmoglobinuric fever is found both in man and cattle, and that in both it may relapse out of season and when far removed from the locality of infection. But the geographical distribution of blackwater fever and redwater fever, which is the same in tropical and sub-tropical climes, seems to part company in the higher latitudes. I believe this difference to be only apparent and due to the fact that in Northern Europe and in Northern America the hæmoglobinuric fever of man is called *paroxysmal hæmoglobinuria* and *epidemic* or *infantile hæmoglobinuria* when it breaks out amongst the infants of a lying-in hospital as in the cases described by Winckleman. I know full well that so-called paroxysmal hæmoglobinuria in northern latitudes is far from being so severe and fatal as the hæmoglobinuric fever of Africa, but then we must remember that in Africa the disease usually attacks those who have been already seriously affected by tropical malaria. The death-rate of blackwater fever varies greatly in different regions. In the Greek Archipelago it is only 22 per cent, in British Central Africa it is 60 per cent. It seems to me therefore that paroxysmal hæmoglobinuria stands to blackwater fever in the same relation as our dysentery (enterocolitis of some authors) to tropical dysentery.

I do not wish to infer that all cases of hæmoglobinuria are parasitic, because mineral poisons (chlorate of potash, etc.), vegetable poisons (some mushrooms), and animal poisons (snake-venom) may induce it, but I certainly believe that most cases of so-called paroxysmal hæmoglobinuria or hæmoglobinuria *a frigore* are parasitic.

I daresay that in a disease with so wide a geographical and zoological distribution we may find several varieties of parasites, but however this may be I consider that there is sufficient evidence to show that blackwater fever is a distinct morbid entity, that it is caused by a specific parasite, that this parasite is an hematozoon belonging to the class of protozoa, that if not identical with *pirosoma bigeminum* it certainly is closely allied to it.

I believe that the parasites of blackwater fever, like those of the æstivo-autumnal group, are to be found chiefly in the capillaries of the internal organs and only sparingly in the peripheral circulation during the initial stage of the paroxysm when the patient experiences feelings of chilliness. Woldert says he found the parasites in about 2 per cent of red corpuscles. This is the case in Texas fever. *Pirosoma bigeminum* may be found in 80 per cent of corpuscles within the capillaries of the liver and spleen, but in only 1, 2, or at most 10 per cent of corpuscles in the peripheral circulation.

The protozoa of "malarial diseases," and those of hæmoglobinuric fever in man and cattle differ greatly in their action on the red-blood corpuscles, but there seems to be a much more important difference between them in their mode of division. The parasites of tertian, quartan, and æstivo-autumnal fevers propagate by means of a multiple rosette-like segmentation, those of hæmoglobinuric fever seem to multiply only by simple binary fission. No other form of division has as yet been witnessed in Texas fever, and both in Texas fever and blackwater fever the parasites are often found in pairs and sometimes still connected.

DIAGNOSIS

From Malaria.—Blackwater fever is usually ushered in by a severe and prolonged *rigor*, and the chill may recur twice or three times in the same day. In tertian and quartan fevers we may also find a severe rigor; but it is far less marked in æstivo-autumnal fever, which is the infection supposed to give rise to hæmoglobinuria.

The *vomiting* in malaria seldom assumes that persistent and grave character which is so characteristic of blackwater fever.

In malarial fevers there is often a a slight sallow colour of the *skin*, but never that sudden, intense saffron or greenish-yellow discoloration which is constantly found in all cases of blackwater fever, not excluding the mildest relapses.

The *urine* of blackwater fever is absolutely pathognomonic, becoming usually as black as porter from the presence of hæmoglobin. In malarial diseases it may be highly coloured from an increased quantity of urobilin, as in any other fever; it may even contain blood in the rare cases in which there be a complicating nephritis.

The *temperature* in blackwater fever is usually low, and several relapses have been recorded in which there was hardly any pyrexia at all. Further, the paroxysms of blackwater fever are so very irregular that out of fifty cases it would be difficult to find two temperature curves exactly alike.

Other peculiar symptoms of blackwater fever are the suppression of urine, the enlargement and tenderness of the liver, the pain over the loins, and the marked leucocytosis.

In malarial diseases there is seldom any difficulty in finding the *specific parasites* within the peripheral circulation—in blackwater fever competent observers have repeatedly failed to find one single parasite.

From Yellow Fever.—The two diseases are co-endemic in the West Indies and in other places. In symptoms and post-mortem appearances they may be so much alike that frequently it may be almost impossible to distinguish between them.

Black vomit is pathognomonic of yellow fever and very rare in blackwater fever.

Black urine is pathognomonic of blackwater fever and very rare in yellow fever.

The *urine* of blackwater fever is dark from the presence of hæmoglobin; in boiling it forms a coagulum of globulin which tends to float; it very seldom contains normally-shaped blood corpuscles. The urine of yellow fever is never dark in the early stages of the disease, but it may become black from the admixture of blood in the late hæmorrhagic period of the disease. The pathognomonic character of the urine in yellow fever is the presence of serum-albumen, which is constantly found, no matter how pale the colour of the urine.

Yellow *discoloration of the skin* is an initial symptom in blackwater fever; it appears late or may be absent in yellow fever.

In blackwater fever the spleen and liver are enlarged and tender to pressure; in yellow fever they are not. The vomit during the first period of yellow fever is usually colourless or somewhat yellowish; in blackwater fever it is from the first of an intense green colour.

The course of yellow fever is constantly the same, and relapses very seldom occur; that of blackwater fever is most irregular, and relapses are the rule.

PROPHYLAXIS

The accurate investigation of the peculiar topographical distribution and seasonal prevalence of the disease may yet lead to important discoveries bearing upon the use of prophylactic measures. To arrive at a knowledge of the topographical distribution and seasonal prevalence peculiar to each locality it is necessary to register only *first attacks*, and obtain from each patient minute information as to his whereabouts in the two or three months previous to the attack. This period will certainly cover the longest incubation period of the disease. Meanwhile we may state in a general way that swampy districts should be avoided during the hot season after the rains.

The prophylactic use of quinine has been strongly recommended by Doering. He believes that quinine not only wards off repeated malarial attacks and thus minimises the chances of blackwater fever, but that the "irritation which quinine exercises upon the red blood corpuscles during the paroxysm is much less when the drug has been constantly administered during the afebrile period."

The daily use of quinine, for a long period, may be injurious, and should therefore be avoided when unnecessary.

TREATMENT

We have as yet no specific remedy against blackwater fever. The use of various styptics, emetics, diaphoretics, and diuretics have each in turn been credited with very beneficial action but on insufficient grounds. Numberless drugs have been tried, but they have proved to be useless or harmful. This employment has been generally empirical or based on erroneous theories as to the pathology of the disease.

Regarding the use of quinine and judging purely from the evidence of facts, we find:—

1. That quinine has no specific action in blackwater fever.
2. That in most cases it is decidedly injurious.

Bastianelli, who accepts both the malarial and quinine theories of blackwater fever, suggests that the drug should be administered only in those cases in which malarial parasites are found in the blood.

So much evidence has been brought forward to prove the injurious actions of quinine in blackwater fever that I think its use should be forthwith discontinued in the treatment of this disease.

If after an attack of blackwater fever ordinary malarial paroxysms supervened without giving rise to hæmoglobinuria, quinine should certainly be administered, because if blackwater fever infection had still been lurking in the system it would have reappeared undoubtedly under the influence of the malarial paroxysms.

We have as yet no remedy known to be of the slightest value in blackwater fever, and the practitioner should be content to treat the disease by careful nursing and ordinary hygienic measures until a specific treatment be sanctioned by laboratory experimentation. We are at present in this same position with regard to typhoid fever and many other infectious diseases.

A moderate dose of calomel (5 grains) to clear the primæ viæ and check the formation of products of decomposition in the digestive tract may be of service. Possibly in most cases an irrigation of the colon is preferable.

The patient should be allowed to drink distilled water freely—we have no better diuretic, and it is necessary to keep the kidneys flushed so that the debris of destroyed blood-corpuscles is washed out from the tubules Warm alkaline drinks will also serve as an emetic to the great quantity of bile which is secreted during the paroxysm, and will thus greatly relieve the persistent and exhausting retching.

The patient should be carefully sponged with tepid water, and this should be done with as little disturbance as possible. Hot fomentations and turpentine stupes may be applied to the loins to alleviate pain and prevent suppression of urine.

The food should be liquid, consisting chiefly of milk and broths. Barley-water, oatmeal-water, lemonade, and the juice of oranges may be given freely. Alcohol is not necessary in all cases, but it should be given when the weakness is marked and the pulse is failing.

The patient should be confined to his bed, and kept in the recumbent position until convalescence is well established.

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Bladder, Injuries and Diseases of.

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MALFORMATIONS

Absence of the bladder is of very rare occurrence, the ureters under these circumstances opening into the urethra, vagina, rectum, or externally on the abdominal wall.

Supernumerary bladders are almost as uncommon, most of the cases described as such being instances of sacculated bladders, the result of irregular dilatation from long-continued obstruction to the escape of the urine.

Ectopion vesicæ, or *extroversion of the bladder*, are the terms applied to a congenital malformation occurring much more frequently in boys than in girls, viz. in the proportion of about 10 to 1, in which, owing to arrest of development, there is an absence of the anterior wall of the bladder, as well as of the corresponding portion of the abdominal parietes, the pubic bones being also separated by a wide interval. Under these circumstances, the mucous membrane of the posterior wall of the bladder, on which the orifices of the ureters can be detected, forms a red vascular projection, being pushed forward by the intestines which lie behind it. In complete cases, this condition, which is accompanied by a constant dribbling of urine from the exposed surface, is associated with absence of the umbilicus, imperfect development of the penis, epispadias, and a bifid scrotum; the testes, which are usually small and undeveloped, are often retained in the inguinal canal. Though the subjects of this deformity are occasionally of a good constitution and live to old age, they are more commonly somewhat weakly, their health suffering from the irritation caused by the constant dribbling away of the urine. The consequence is, that a large proportion succumb in early life to some intercurrent disease, *e.g.*, inflammatory affections of the skin and parts around, sometimes of an erysipelatous nature; or secondary renal disease, from septic inflammation spreading upwards from the exposed mucous surface, and causing suppurative pyelitis or nephritis.

Treatment.—Various operations have been devised with the object of protecting the exposed mucous membrane, and providing a cavity in which the urine may be retained. These are as follows:—

1. Covering in the mucous surface by turning over skin-flaps taken from the abdominal wall and groin (Wood). By this method the mucous surface can be closed in, except at its lower part, where it is continuous with the open groove on the dorsal surface of the penis, which represents the urethra, and the urine is therefore more easily collected in some form of apparatus. An objection to this plan is, that the hairs on the under surface of the skin-flaps continue to grow, and often become encrusted with deposits of phosphates from the urine, which, under these circumstances, is usually alkaline; distinct calculi sometimes form, causing considerable irritation, and occasionally requiring removal. To prevent this complication arising, it is recommended to destroy the hair-bulbs by electrolysis or by caustics previous to operation.

2. Closing in the bladder by suturing together the margins of the mucous membrane. Flaps of mucous membrane are dissected up from either side, from without inwards, *i.e.* from their line of junction with the skin, then turned inwards and united by sutures in the middle line. To allow of their approximation, and at the same time to bring together the separated pubic bones, the sacro-iliac synchondrosis should previously have been divided on either side (Trendelenburg). Another method is to cover in the closed bladder by two marginal flaps consisting of skin only (Milton, Anderson); or, also including the recti muscles and a portion of the pubic crest from either side (Mikulicz), tension being relieved by two lateral incisions carried more externally.

3. Converting the mucous membrane into a cutaneous surface, or one consisting of cicatricial tissue. This method, which is effected by dissecting off the mucous membrane, and afterwards skin-grafting, or by destroying it with caustic, is liable to be followed by troublesome contraction of the orifices of the ureters. To prevent this complication, the mucous membrane may be freely separated round the orifices of the ureters, which are attached to the root of the penis (Sonnenburg).

4. Diverting the stream of urine from the open bladder: (*a*) by establishing a fistulous communication between both ureters and the rectum or colon; (*b*) by attaching one ureter to an opening in the loin, and removing the opposite kidney, so as to avoid the inconvenience of a double fistula in the lumbar region (R. Harrison). The former method is attended by the risk of septic inflammation—from the decomposition of the urine mixing with fæces, and being retained in a closed cavity—spreading up along the ureter to the kidney. In the latter there is the tendency to contraction of the orifice of the ureter in the loin, as well as the inconvenience of a fistulous opening in this situation; moreover, the operation of removal of one kidney is in itself always a serious procedure.

5. Forming a communication between the bladder and the colon (cystocolostomy). The mucous surface of the bladder is freely separated, and its margins are then attached by sutures to the margins of an opening in the sigmoid flexure

of the colon, the result being that the urine then passes entirely by the bowel. (Tuffier).

The results of the different operations are not in many cases very satisfactory, for even when the exposed mucous membrane is successfully covered in by flaps of skin, it is generally necessary for the patient to wear an apparatus to collect the urine, which still escapes, owing to the absence of a sphincter muscle at the neck of the cavity which represents the bladder.

MALPOSITIONS.—*Hernia or Cystocele.*—A protrusion of the bladder may take place in the median line above the pubes, when the abdominal wall is weakened from any cause, as for example after laparotomy. In females who have borne children it not uncommonly protrudes into the vagina, owing to yielding of the anterior wall of the passage, sometimes projecting beyond the vulva (*vide* "Vaginal Cystocele"). In rare cases the bladder may form part of the contents of an inguinal or femoral hernia, and instances are recorded where it has protruded through the obturator or ischiatic foramina. Unless of large size the protruding portion of the bladder is generally uncovered by peritoneum. The diagnosis of an inguinal or femoral cystocele is always a difficult matter, and the condition is often not recognised until the bladder is exposed—and sometimes accidentally opened—in an operation for the relief of symptoms believed to be those of an ordinary strangulated hernia. On inquiry it will, however, generally be found that there were, in addition to the usual symptoms, pain, referred especially to the neck of the bladder, with increased frequency in micturition, and perhaps small quantities of blood in the urine.

Treatment.—When the protrusion occurs in the median line, a well-fitting abdominal belt should be worn. In the inguinal and femoral varieties a truss should be applied, or an attempt may be made to close the opening, after the protrusion has been reduced, by an operation somewhat similar to that performed for the radical cure of hernia.

Inversion of the bladder is the term applied to a condition rarely met with in female children, in which, as the result of straining during micturition, a prolapse of the bladder takes place through the urethra in the form of a red vascular mass, or pyriform tumour, which may project beyond the labia. The protrusion involves the entire thickness of the vesical wall, and must therefore be distinguished from a prolapse of relaxed mucous membrane into or through the urethra, a condition which is not very uncommon in women. To produce contraction of the dilated urethra and neck of the bladder, the interior of the canal should be cauterised after the prolapse has been reduced.

INJURIES

RUPTURE OF THE BLADDER is most likely to occur when the viscus is distended with urine, for if empty it lies under cover of, and is protected by, the pelvic bones. It may be produced in various ways, usually as the result of injury, for example by a blow or crush involving the lower part of the abdomen; it frequently complicates cases of fractured pelvis, the bladder being ruptured by the same force, or penetrated by a fragment of bone. Though in exceptional cases it may occur from simple over-distension without any external violence, it will usually be found that under these circumstances the bladder-wall was previously the seat of ulceration, or thinned and weakened from disease.

Rupture of the bladder may be divided into intra-peritoneal and extra-peritoneal according to the situation of the rent in its wall.

(*a*) *In intra-peritoneal rupture*, the most common variety, the posterior or upper wall of the bladder, *i.e.*, the portion invested by peritoneum, gives way, and the peritoneal cavity being opened, extravasation of urine usually takes place into its interior. The commonest situation is the posterior wall, the rent usually running in a more or less vertical direction, and when due to injury, often for a distance of one or two inches; in idiopathic cases the opening is of small size, and generally more circular in shape. The urine may become extravasated widely through the lower part of the abdominal cavity, or it may be limited by coils of intestines, sometimes forming a more or less defined swelling above the pubes, or behind the bladder, and then perceptible on examination by the rectum. Occasionally the rupture is at first subperitoneal, the urine collecting between and separating the muscular and serous coats, and subsequently making its way through the peritoneum into the abdominal cavity.

The symptoms under these circumstances are usually of a well-marked character. There is severe collapse, with burning pain in the lower part of the abdomen, and often a constant desire to micturate, with inability to do so, though at times a small quantity of almost pure blood may be expelled from the urethra. A catheter can be introduced into the bladder without difficulty, and it will be found either quite empty, or perhaps a little bloody urine can be drawn off. Occasionally, a considerable amount of fluid tinged with blood can be evacuated, owing to the fact that the end of the instrument passes through the rent in the bladder-wall into the peritoneal cavity and draws off urine mixed with peritoneal fluid and blood. Under these circumstances the movement of the catheter, which was restricted so long as it remained in the empty bladder, will be found unusually free, and its end can often be felt through the abdominal wall much more distinctly than would be the case if it was in the interior of the bladder. Inquiry should always be made whether the bladder was full at the time of the injury, for if so, and it is afterwards found empty, this is presumptive evidence of rupture having taken place. In cases where the diagnosis is doubtful a measured amount (10-12 oz.) of boric lotion may be injected into the bladder and then allowed to run out. If the bladder becomes distended, and the same quantity afterwards escapes, there is no rupture; but if only a small proportion of the lotion returns, it is probably because some of the fluid has passed through a rent in the bladder into the peritoneal cavity. It has also been suggested in doubtful cases to inflate the viscus with hydrogen or with air; if it is intact, a localised tympanitic tumour, taking the form of the distended bladder, will be apparent; if ruptured, the gas or air passing into the peritoneal cavity will cause general tympanites.

If the injury does not rapidly prove fatal, and no treatment is adopted, symptoms of peritonitis usually supervene, and in most cases death soon takes place.

(*b*) *In extra-peritoneal rupture* the anterior wall or base of the bladder, *i.e.* the part uncovered by peritoneum, gives way, and the peritoneal cavity not being opened, the urine escapes into the pelvic cellular tissue in front of or beneath and behind the bladder. It may infiltrate the parts widely, or collect and form a localised swelling above the pubes, or between the bladder and rectum, sometimes in the middle line, sometimes more on one side than the other. Occasionally the urine travels upwards for some distance in the subserous areolar tissue, *e.g.* as high as the umbilicus, and then bursts through the peritoneum into the abdominal cavity; or it tracks downwards along the inguinal canal to the scrotum, or through the obturator foramen and femoral canal to the upper part of the thigh, forming a diffused swelling in these situations.

The general symptoms are very similar to those of intra-peritoneal rupture, and in many cases a certain diagnosis cannot be made without an exploratory incision. In this injury, however, the end of the catheter, which will not pass into the peritoneal cavity, can occasionally be felt outside the bladder in the cellular tissue between it and the rectum. A diffused swelling is sometimes apparent above the pubes or in the perineum, owing to the extravasation of urine into the cellular tissue in one or other of these situations.

Unless an outlet is speedily provided for the urine, pelvic cellulitis, often followed by peritonitis, is set up, and the case soon terminates fatally.

Diagnosis.—Except in cases where the rent is small, and either valvular or blocked by a coil of intestine, when many of the characteristic symptoms of rupture may be absent, the diagnosis of this injury is not usually very difficult. Ruptured urethra may be distinguished by the fact that it will be difficult or impossible to introduce a catheter into the bladder, the point of the instrument being arrested at the seat of laceration; if, however, it can be passed, a quantity of clear urine can usually be drawn off. In most cases hæmorrhage takes place from the urethra independently of any attempt at micturition; unless relief is afforded, evidences of distension of the bladder soon appear.

Treatment.—In intra-peritoneal rupture the indication is to open the abdomen in the middle line, close the rent in the bladder-wall, and thoroughly cleanse the peritoneal cavity. In closing the opening in the bladder, Lembert's method of suturing should be employed, the peritoneal surfaces of the margins of the wound being inverted and brought into contact by fine silk sutures, which should transfix the serous and muscular coats, but not include the mucous membrane. To make sure that the bladder is water-tight it should be tested by injecting boric lotion through the urethra; if any escapes through the wound, additional sutures should be inserted at the weak spot. The peritoneal cavity is then thoroughly irrigated, and all blood and fluid afterwards removed. The external wound may then be completely closed, or a glass drainage tube may be inserted, reaching to the recto-vesical cul-de-sac, so that if there is any leakage of urine there may be an outlet for it. To prevent this accident, which may occur if the viscus becomes distended with urine, a catheter should be retained in the bladder or passed at regular intervals for several days.

In extra-peritoneal rupture free incisions should be made in the perineum or above the pubes to provide an outlet for the extravasated urine, and one or more drainage-tubes should be inserted. If the rent in the bladder-wall is accessible it should be closed by sutures, which should include only the muscular coat.

The prognosis after operation is much more favourable in this injury than in intra-peritoneal rupture, where the mortality is very high, even after suture of the bladder. In both cases the result will be influenced by the interval which elapses between the injury and the performance of the operation, for if there has been much delay, and especially if the urine is in an unhealthy condition, peritonitis or cellulitis will probably result, followed in most cases by death.

PENETRATING WOUNDS OF THE BLADDER are sometimes met with as the result of gunshot or other injuries, *e.g.* stabs involving the lower part of the abdomen, falls upon some sharp object, which penetrates the perineum, etc. The general symptoms will be very similar to those of rupture of the bladder, but in addition there will often be an escape of urine mixed with blood from the external wound, which renders the diagnosis more easy. It sometimes happens that the bladder is accidentally opened in operations for the removal of ovarian or other forms of abdominal tumour, owing to the presence of adhesions. The treatment of this injury will be the same as in cases of rupture, and when occurring during the performance of an operation, the wound in the bladder should at once be closed by sutures.

FOREIGN BODIES

Foreign bodies of various kinds may gain access into the interior of the bladder in several ways, viz. :—

1. ALONG THE URETHRA.—This is the usual route, it occasionally happening that pieces of gum elastic catheters are broken off and left in the bladder during catheterism, or when tied in and retained there, and the same accident may occur with bougies in the treatment of stricture. Not infrequently portions of pencil, penholders, bodkins, hairpins, etc., introduced into the urethra by the patients themselves to allay some irritation or for sensual purposes, slip out of reach, and then travelling backwards along the canal, make their way into the bladder, where they remain.

2. THROUGH THE BLADDER-WALL.—In other cases a piece of bone from a diseased or fractured pelvis; a foreign body from the intestine or vagina (*e.g.* a pessary); the contents of a dermoid cyst; portions of a fœtus in cases of extra-uterine gestation; hydatid cysts forming in the pelvis—may ulcerate through the bladder-wall, and make their way into its interior; in rare cases of gunshot injury, a bullet or piece of clothing may penetrate the bladder and remain there.

3. ALONG THE URETER.—Hydatid cysts forming in the pelvis of the kidney, or foreign bodies (*e.g.* bullets) which have lodged there, may in rare cases make their way downwards along the ureter into the bladder.

Symptoms.—Though a foreign body may be present in this situation for a considerable time without causing any definite symptoms, it more frequently sets up irritation, micturition becoming painful and frequent, and traces of blood appearing in the urine. Cystitis soon supervenes, and the urine becoming alkaline, the foreign body becomes encrusted with phosphates, forming the nucleus for a stone, and giving rise to all the symptoms of that condition.

Treatment.—When, as sometimes happens, there is a doubt as to the presence or not of a foreign body, the cystoscope will prove of service, for it will not only reveal it, if one is there, but it will also show its exact position, and in this way often facilitate attempts to remove it. When the foreign body is a piece of catheter, bougie, pencil, etc., it can often be seized by a lithotrite, or by a special forceps, so constructed that it is caught between the blades in its long axis. If hard and brittle, its extraction will sometimes be rendered more easy, if it is first broken up by crushing it with a lithotrite. If, however, this is impracticable, and the patient is a male, perineal or suprapubic cystotomy must then be performed, and the same treatment will usually be required when the foreign body consists of part of the contents of a dermoid or hydatid cyst, etc. In the case of females, most foreign bodies can be removed through the dilated urethra.

DISEASES

INFLAMMATION OR CYSTITIS.—This is usually met with as a chronic, much less frequently as an acute affection, and in the majority of cases is due to the introduction of micro-organisms from without. Their presence in the urine is not, however, necessarily accompanied by inflammation, for if the bladder is healthy and uninjured, and there is no obstruction to the escape of its contents, its walls may remain free from infection, and no formation of pus takes place, this condition being known as *bacteriuria*. If, however, organisms are present in the urine, and the mucous membrane of the bladder becomes injured or irritated in any way, or if there is some cause of obstruction, and retention of urine results, then bacterial infection of the vesical walls occurs, and pus forms, the condition being then termed *suppurative cystitis*. In cases where the vesical infection is superficial and slight, and the urine, though containing bacteria, remains almost free from pus, the condition is sometimes described as *catarrhal cystitis*, and may be regarded as a stage intermediate between bacteriuria and suppurative cystitis. *Simple cystitis* is the term applied by some to a condition, which is regarded as a stage preliminary to that just described, where the inflammation is due to some mechanical or chemical irritant, *e.g.* a calculus, uric acid or oxalate of lime crystals, or the active principle of cantharides, etc., present in the urine. It differs from the preceding in the fact that the urine is free from both pus and micro-organisms, but it contains blood-corpuscles and numerous epithelial cells. Examined with the cystoscope, the appearance presented is one of extreme congestion of the mucous membrane, very similar to that described under *hæmorrhagic cystitis*. The inflammation may subside at this stage, but often runs on to the suppurative form, pus and organisms then being found in the urine, and bacterial infection of the bladder-wall also taking place.

ETIOLOGY.—As obstruction to the escape of the urine predisposes to cystitis, this explains why it so frequently complicates cases of urethral stricture and enlarged prostate, being especially liable to occur in the latter condition when a quantity of residual urine is habitually present in the bladder. In cases of disease or injury of the spinal cord, in addition to trophic changes in the bladder from disordered innervation, the loss of power in its muscular coat by favouring retention again acts in the same way. Any local cause of irritation, by causing congestion of the bladder, also acts as a predisposing cause, *e.g.* stone, tumours, foreign bodies, retained catheters, the introduction of any instrument, etc.

The exciting cause of suppurative cystitis is generally septic infection, from the introduction of micro-organisms, which may gain admission into the bladder either directly or indirectly.

DIRECT INFECTION MAY TAKE PLACE IN VARIOUS WAYS, most commonly through the urethra, less frequently through the ureter, and rarely through a breach in the bladder-wall.

Infection through the urethra is often caused by the passage of a septic catheter; in cases where a sterilised catheter or sound is employed, it may be due to the instrument pushing on, or carrying with it, into the bladder, the organisms which are normally present in the healthy urethra, and especially when it is in any way inflamed or affected with disease; *e.g.* in cases of stricture, organisms are commonly found in the recesses or dilatations in the walls of the urethra behind the constriction, so that even if the meatus and anterior part of the canal are disinfected, they are still liable to be carried on into the bladder by any instrument. In urethritis from any cause, whether gonorrhœal or due to spread of septic inflammation from the glans penis, vulva, or vagina, etc., spontaneous infection of the bladder may take place, independently of any instrumentation, from extension backwards along the urethra. The same result, viz. infection through the urethra, may also occur without the existence of any urethritis, for in cases of incontinence, especially when due to overflow from distension, the organisms which are normally met with about the external genitals and anal regions, readily work their way upwards to the bladder in the stream of urine which is continually oozing from the orifice of the urethra.

Infection through the ureters may happen when the kidneys themselves are the seat of septic disease, or in cases of general septicæmia, the bladder becoming infected by organisms, which reach it through the urine. This mode is sometimes described as "renal," or "descending infection" of the bladder.

Infection through a breach in the bladder-wall, e.g. in cases of vesical fistula, abscesses opening into the bladder, penetrating wounds, etc., only rarely takes place.

Indirect Infection of the Bladder through the blood-vessels, or more commonly through the lymphatics, may occur in cases of septic disease involving the parts in its neighbourhood. There are also good grounds for believing that in some cases organisms (*e.g.* the bacillus coli) may be transmitted directly from the rectum to the bladder through the medium of the lymphatics. In this mode of indirect infection, contrary to what happens in the preceding, the bladder-wall becomes first involved, and the urine secondarily.

The micro-organisms found in the urine may be divided into two classes, according as they do or do not possess the power of decomposing urea, the former being the most injurious. The bacillus coli is the one most frequently present in the urine in cases of bacteriuria, and also in suppurative cystitis when the fluid is acid, its power of decomposing urea—if it possesses it—being very slight, and not exercised to any extent while in the bladder. The urobacillus liquefaciens septicus (the proteus of Hauser) is the organism usually found when the urine is ammoniacal; it quickly decomposes urea, and when present in acid urine, the reaction becomes alkaline and the odour ammoniacal within a few hours. The staphylococcus pyogenes and the streptococcus pyogenes are present in a large proportion of cases, rarely by themselves, almost always associated with one or both of the preceding varieties; the former readily decomposes urea, while the latter does not possess this property (Moullin). In addition to the ordinary pyogenic micro-organisms, certain specific organisms are sometimes found to be present, usually in association with them, less frequently acting by themselves, *e.g.* the gonococcus in gonorrhœal cystitis, the typhoid bacillus in the cystitis which occasionally occurs in the course of typhoid fever, and the tubercle bacillus in cases of tubercular cystitis.

Acute Cystitis

Pathology.—In acute cases there is intense congestion of the vesical mucous membrane, most marked about the trigone and neck of the bladder. Examined with the cystoscope in the early stage, the bladder-wall appears of a bright red colour, the injected vessels being plainly visible as branching lines, and in places small patches of extravasated blood can be distinguished. As the inflammation advances the colour changes to a dark red or purple, the outlines of the vessels becoming lost, and the mucous membrane assuming a swollen velvety appearance. In severe cases the dilated vessels give way, and profuse hæmaturia may occur, the condition being then sometimes described as "hæmorrhagic cystitis." If it progresses beyond this stage, the epithelial layer may become detached, and superficial ulceration takes place, pus forming on the surface and sometimes in the walls of the bladder. Examined microscopically, micro-organisms will be found in abundance in the mucous and submucous coats between the proliferating epithelial cells, and in severe cases they may extend more deeply, even infiltrating the muscular coat.

MEMBRANOUS, OR EXFOLIATING, CYSTITIS are the terms applied to a form of acute inflammation of the bladder, accompanied by a fibrinous exudation upon, or into and beneath, the mucous membrane. Exfoliation of the false membrane takes place, the exudation separating and coming away in shreds or sloughs, which are expelled with the urine. The sloughs may be quite superficial, or extend deeply, involving a portion or the whole of the thickness of the muscular coat; occasionally a complete cast of the interior of the bladder becomes detached. This condition, which always indicates an intense form of cystitis, is most frequently met with in females after difficult labours, where, in addition to septic infection of the urine, there has also been retention. It may also occur in males in cystitis secondary to injury or disease of the spine, as well as in severe cases of prostatic disease.

SYMPTOMS.—There is acute pain in the perineum and region of the bladder, with tenderness on pressure above the pubes. The bladder is extremely irritable, micturition being very frequent, and accompanied by great pain and straining, especially at the end of the act. The urine, which is passed in small quantities, soon becomes purulent, and neutral or alkaline in reaction. Examined microscopically, in addition to pus the deposit will be found to contain blood-corpuscles, proliferating epithelial cells, and numerous micro-organisms. In "hæmorrhagic cystitis" blood is sometimes present in considerable quantity, the urine assuming a bright red colour, and often containing small clots.

TREATMENT.—The cause of the cystitis having, if possible, been removed, the patient should be confined to bed, and the pain relieved by warm baths, fomentations to the perineum and lower part of the abdomen, and suppositories of morphia (gr. $\frac{1}{4}$ to $\frac{1}{2}$) or belladonna (gr. i.). The bowels should be kept open by saline purgatives, and stimulants should be forbidden. If the urine is acid, alkalies and tinct. hyoscyamus should be given internally; the diet should consist mainly of milk, and demulcent drinks, *e.g.* barley-water should be freely given. During the acute stage all instrumentation and irrigation of the bladder should be avoided, for if attempted it will cause great pain, and tend to aggravate the symptoms. If the urine becomes alkaline, or if it remains so after the acute symptoms have subsided, and pus continues to be present in any quantity, then urinary antiseptics will be required, and the treatment will be the same as that described for chronic cystitis. In the hæmorrhagic form the blood soon disappears from the urine if the patient is kept at rest in bed, and some hæmostatic is given internally.

For severe cases of acute cystitis, *e.g.* when secondary to advanced prostatic disease, cystotomy and bladder drainage may be required, as in the chronic form. The same treatment is often advisable in membranous cystitis, especially when occurring in males, as it allows of the removal of the false membrane, portions of which, when detached, are liable to block the vesical orifice of the urethra and cause retention of urine.

CHRONIC CYSTITIS

Pathology.—The mucous membrane becomes thickened and altered in appearance, assuming a dark slate colour, and presenting patches of ecchymosis or superficial ulcerations, which may be coated over with muco-pus or deposits of phosphates; sometimes it is rough and lined with a layer of villous-like granulations, which readily bleed. Occasionally it presents a "nodular" appearance, the lymphatic foci, which are normally present in the mucous membrane, becoming enlarged, and developing into prominent and vascular nodules. In cases of long standing, and when the cystitis is secondary to some cause of obstruction, *e.g.* stricture, the bladder often becomes contracted and its muscular coat hypertrophied, thick bands standing out prominently beneath the mucous lining, and interlacing with one another in different directions. Irregular dilatation, or "sacculation," is often associated with this condition, so that pouches and depressions are produced between the fasciculi, in which urine lodges and often decomposes, or calculi may form and sometimes become encysted. As the result of thinning and ulceration of the walls of these pouches, perforation may occur, followed by extravasation of urine, a complication which is usually fatal. In some cases the bladder may be dilated, and its walls generally are much thinned; this condition, the reverse of that just described, is usually found associated with prostatic hypertrophy, passive distension of the viscus taking place as the result of long-continued retention of urine.

The *SYMPTOMS* are of a similar nature to those met with in acute cystitis, though much less severe, the most prominent being an irritable condition of the bladder, with an altered state of the urine. Micturition is increased in frequency, and attended by more or less pain. The urine soon becomes purulent and alkaline in reaction, giving off in severe cases an offensive ammoniacal odour. It is turbid when passed, and deposits a quantity of viscid, ropy mucus, which contains pus, blood, and epithelial cells, with numerous micro-organisms and crystals of triple phosphate and phosphate of lime. In neglected cases, as the disease progresses, there is a gradual loss of flesh and strength; the patient becomes sallow and emaciated, digestion becoming impaired, with a distaste for food. A low form of delirium sets in at night, and symptoms of uræmia frequently supervene, for the inflammation tends to spread upwards along the ureters to the pelves of the kidneys; the result is that the patient, becoming worn out by the pain and frequent calls to pass water by night as well as by day, sooner or later succumbs to septic poisoning or to some secondary renal complication.

DIAGNOSIS.—Cystitis may be simulated by *certain functional disorders* of the bladder of nervous origin, which are often attended by pain (cystalgia) and increased frequency in micturition, the latter condition being sometimes described as "irritable bladder." They may, however, be distinguished by the fact that the urine is healthy and free from pus, and there is no evidence of the existence of any of the causes which usually give rise to cystitis. The patient is often of a nervous temperament, or some lesion will be found in the neighbouring parts, *e.g.* uterine disease, hæmorrhoids, fissure of the anus, phimosis, a contracted urethral meatus, etc., the bladder symptoms under these circumstances being due to reflex irritation. In other cases a similar condition may be due to the direct irritation of urine containing sugar, or an excess either of uric acid or phosphates, the symptoms quickly subsiding if it is restored to a healthy condition. *Pyelitis* is another affection which must be distinguished from cystitis, for it is accompanied by the presence of pus in the urine, and often by pain and increased frequency in micturition. In pyelitis, however, the kidney is frequently somewhat tender upon pressure, and occasionally a slight fulness can be detected upon palpation; the urine retains its acid reaction for a considerable time, and the pus, which is often present in considerable quantity, is more intimately diffused, and does not so quickly separate and settle at the bottom of the vessel as when it is derived from the bladder. In doubtful cases a cystoscopic examination of the interior of the bladder, or examination of a specimen of urine obtained before it enters the bladder by catheterisation of the ureters, will sometimes show whether the pus is of renal or vesical origin. In most cases of cystitis some cause will be apparent, *e.g.* the irritation of a stone or a new growth, prostatic enlargement, urethral stricture, etc., or there will be a history of septic infection of the bladder having been caused in one or other of the ways described. In the absence of any definite cause, and especially if the patient is a young adult, the cystitis is often due to tuberculosis, and the possibility of the existence of a "solitary ulcer" should also be borne in mind.

TREATMENT.—The first indication is to remove, if possible, the cause of the cystitis, and this having been effected, to correct the septic condition of the urine. If secondary to a stricture of the urethra, this should be dilated or divided; if calculi, foreign bodies, or growths are present in the bladder, they should be removed; if due to the constant irritation of residual urine in enlargement of the prostate, the bladder should be regularly emptied by a catheter, and the treatment appropriate for the prostatic disease should also be followed out.

To correct the septic condition of the urine, urinary antiseptics, *i.e.* drugs which exert a germicidal action upon the organisms present in the urine, should be employed, and these may be administered by the mouth or introduced directly into the bladder. The antiseptics usually given internally are ***boric acid, salol, benzoic acid, naphthalin, benzonaphthol***, and the ***benzoates of soda*** and ***ammonia***, but inasmuch as they act upon the bladder through the medium of the urine, and therefore require to be first eliminated by the kidneys, the germicidal power which they excite upon the organisms present in the bladder is but slight. Moreover, they have to be given in fairly large doses, continued for some length of time, and the result is that the digestive functions of the patient are very liable to become impaired. Though their action, therefore, is not very powerful, still they form useful adjuncts to other methods of treatment, and their administration is usually attended by benefit. Another class of drugs which often prove of service in cases where pus and mucus continue to be present in the urine after the irritation has subsided, are those which stimulate directly the vesical mucous membrane through the urine, *e.g.* ***oil of sandal wood, turpentine, cubebs, copaiba, cantharides***, etc. When pain and irritability of the bladder are prominent symptoms, ***morphia*** or ***belladonna suppositories*** may be used, and *tinct. hyosc.* given internally. A useful mixture in many cases of chronic cystitis is one containing tinct. hyosc. (m. 30), boric acid (gr. 10), and inf. buchu. (1 oz.), to be taken three or four times a day.

As in the acute form of the disease, alcohol should be avoided, and fluids should be given freely, *e.g.* several pints of distilled water to drink daily. If salol or some other antiseptic is also given by the mouth, the urinary tract is constantly washed out from above downwards with a large quantity of a slightly antiseptic fluid. "The urine becomes exceedingly diluted, which circumstance diminishes its nutritive value for microbes, while the salol renders it still more adverse to bacteria" (Rovsing). When the urine is acid, and especially if it is concentrated, depositing urates, as often happens in gouty subjects, then ***alkalies*** should be administered, *e.g.* potassæ bicarb. or cit., with plenty of fluids; and alkaline mineral waters (potash or lithia) should also be freely given.

Vesical Irrigation.—The most effectual method of treating chronic cystitis is by ***vesical irrigation***, *i.e.* washing out the interior of the bladder with an antiseptic lotion. A flexible catheter, connected with a glass funnel by a piece of india-rubber tubing two or three feet in length, is passed into the bladder. The funnel is raised above the level of the patient's body in the recumbent position, and filled with fluid, which is allowed to run slowly into the bladder until it begins to cause a feeling of fulness or discomfort. The funnel is then lowered below the patient's body and held over a vessel, into which the fluid runs out of the bladder on the syphon principle. This process is repeated several times until a pint or more of fluid has been allowed to run in and out of the bladder.[1] The amount of fluid which the bladder will comfortably hold depends upon its capacity and the degree of inflammation present, often not exceeding three to six oz. This process should be repeated once or twice a day, the temperature of the fluid being about 100° F., care being taken that the catheter and apparatus used are perfectly aseptic. The solutions employed in this way are various, *e.g.*, boric acid (gr. 5-10 to 1 oz.); perchloride of mercury (1-10,000); nitrate of silver (1-2000); sanitas (1-20); permanganate of potash (1-2000); boro-glycerine (1-20); quiniæ sulph. (gr. 2 to 1 oz.); carbolic acid (1-100). As injections of mercury or silver nitrate often cause pain, it is always advisable to commence with a weak solution, and to gradually increase the strength according as it is borne by the patient. When there is a tendency to phosphatic deposits on the wall of the bladder, dilute lotions of nitric, phosphatic, or acetic acid often act beneficially. In cases where ulceration is present, the introduction of a small quantity of iodoform emulsion (gr. 10 to 1 oz.) is often attended by good results.

Instillation.—When irrigation causes severe pain or excites violent contractions of the bladder, *instillation* should be employed as a substitute, and it will often be well borne and attended by good results. This consists in injecting slowly drop by drop—and allowing to remain in the bladder after it has been emptied of its contents—a small quantity (*e.g.* 20-40 drops) of some antiseptic fluid, of greater strength than is used for irrigation, *e.g.* silver nitrate (1-5 in 100) or perchloride of mercury (1 in 1000 or 1500). When the pain is great, it may be prevented or lessened by the previous injection of a small quantity of a 5 per cent solution of antipyrin or cocain, which act as useful local anæsthetics to the bladder wall.

Bladder-drainage.—In obstinate cases of chronic cystitis, when the symptoms do not yield to the ordinary methods of treatment, and the patient's strength is becoming worn out by the pain and frequent calls to pass water by night as well as by day, operative interference will be required. The indication under these circumstances is to place the bladder temporarily in a state of complete rest, and this is effected by draining it either through a perineal or suprapubic opening; at the same time it can be washed out more effectually through one or other of these routes, and also with much less pain to the patient, than per urethram. Another advantage of opening the bladder is that its interior can be thoroughly explored with the finger, and occasionally a cause for the cystitis, *e.g.* an encysted calculus, a patch of ulceration, or a small growth in the early stage,—the existence of which was previously not suspected, or not detected by cystoscopic or other methods of examination—can be recognised and treated.

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[1] Vesical irrigation may also be performed without the use of a catheter, by the employment of *hydrostatic pressure*. A conical nozzle, which fits the external urinary meatus, is connected by a piece of indiarubber tubing with a vessel containing fluid, raised five to seven feet above the level of the patient's body. The urethra is first flushed out by allowing some of the fluid to enter and then to escape by the side of the nozzle. On compressing the nozzle tightly, so that no fluid escapes, the urethra becomes distended and the sphincter vesicæ soon relaxes, allowing the fluid to enter the bladder. On withdrawing the nozzle, the fluid is expelled from the bladder, often with considerable force.

The bladder may also be irrigated through a catheter introduced per urethram, or by hydrostatic pressure; or by a Higginson's syringe fitted with a conical nozzle, which fits the external urinary meatus.

If the suprapubic operation is selected, it affords the additional advantage of enabling the interior of the bladder to be directly inspected, for if a Fenwick's "caisson" speculum is introduced through the wound, and its lower end gently pressed against the mucous membrane, almost the whole of its surface can be brought into view, by moving the speculum from place to place and throwing a bright light down it, after all fluid has been sucked out or removed with a sponge. If no cause is found, a tube is left in the wound for a few days and then removed. Meanwhile, the urine continually drains away through the opening through which the bladder is regularly washed out, and in the course of three or four weeks the wound generally closes. As the result of the complete physiological rest afforded during this period, it will usually be found that by the time the wound has healed, the inflammation will have subsided and the bladder will be restored to a healthy condition. If, as often happens in cases of long standing, one or more patches of superficial ulceration are found, these should be either cauterised or curetted; if phosphatic deposits are present, they should be scraped away, and in both cases a similar course of drainage should afterwards be adopted.

In certain very severe cases of chronic cystitis of long duration, the bladder may have become so contracted from thickening of its muscular coat, which is rigid and unyielding, and its mucous lining may be so altered in character that it is impossible, even by prolonged drainage, to restore it to a healthy condition. Under these circumstances *it may be necessary to establish a permanent suprapubic or perineal opening*, a silver or vulcanite tube being worn in the wound, and either plugged or connected with a urinal attached to the patient's leg, so that the urine may continually drain away.

In the case of *females*, bladder-drainage is rarely required. It may be carried out through a suprapubic opening, or by an incision into the bladder through the vesico-vaginal septum, the former method being preferable. Simple dilatation of the urethra gives good results in cases of pain and irritability of the bladder due to spasmodic contraction of its neck, but the sphincter muscle soon recovers itself, so that the drainage afforded by this treatment is only partial and, as a rule, very temporary.

Tuberculous Disease of the Bladder

This may be primary, but is more commonly met with as a secondary affection, owing to extension of the same condition from some other part of the genito-urinary tract, *e.g.* the kidneys, prostate, testes, or seminal vesicles; it also occasionally develops in the course of cases of pulmonary tuberculosis. It is more frequent in males than females, occurring more commonly in young adults than in persons advanced in years, and being only occasionally met with in children.

Pathology.—The bladder may become infected with tubercle, either directly through the urine, or indirectly through the blood-vessels and lymphatics. Direct infection through the urine may occur in cases of renal tuberculosis, the bladder-wall becoming inoculated from contact with tubercle bacilli present in the urine. This method of invasion does not occur so long as the bladder-wall is healthy and intact, but if the mucous membrane becomes irritated or injured in any way, *e.g.* by a calculus, or by the introduction of a catheter, sound, etc., then the affected part is very liable to become inoculated from the urine. More frequently, infection takes place indirectly from the kidneys or testes through the medium of the lymphatics. The lymphatics of both these organs communicate with those of the base and neck of the bladder, and this is the situation where tuberculous deposits are usually first found. The bladder-wall may also become invaded by direct extension from the seminal vesicles (which are so often affected in cases of genito-urinary tuberculosis, especially when the disease originates in the testes), less frequently by extension from the prostate.

In the early stage, deposits of tubercle are found in the submucous tissue, chiefly about the trigone and surrounding the orifices of the ureters; after a time these soften and break down, giving rise to the presence of numerous small ulcers, which may subsequently coalesce to form one or more ulcerated surfaces of larger size. The ulceration, which at first is quite superficial, may eventually involve the entire thickness of the bladder-wall, fistulous communications forming between the viscus and the rectum or vagina. If commencing in the bladder, the disease usually extends to other parts of the genito-urinary tract; when involving the bladder secondarily, owing to extension from the genital organs, the infection frequently spreads upwards along the ureter to the kidneys.

Symptoms.—In the early stage the characteristic symptoms are increased frequency in micturition, accompanied by pain and often by the presence of minute quantities of blood in the urine, especially at the termination of the act. Sooner or later cystitis ensues, and all the symptoms characteristic of this condition are then superadded, the urine containing pus, as well as the tubercle bacilli. When ulceration has taken place, pain and irritability of the bladder become more prominent symptoms, and there may also be an increase in the amount of blood present in the urine.

The *prognosis* of this affection when commencing in the bladder is always unfavourable, for though it often runs a somewhat chronic course, with periods of temporary improvement, the tendency is for the disease to progress locally, and also spread to other parts, the subjects of it frequently developing evidences of renal or pulmonary tuberculosis, which prove fatal; or death may be due to exhaustion, the patient being worn out by the severity of the bladder-symptoms.

Diagnosis.—The existence of this condition in the early stage should be suspected in young adults complaining of irritability of the bladder with traces of blood in the urine, for which no evident cause can be found, such as stone, stricture of the urethra, etc. The diagnosis will be confirmed if there is a tuberculous family history, or evidences of tubercle in the lungs or in some other part of the genito-urinary tract; also if the tubercle bacillus can be detected in the urine. A cystoscopic examination will usually reveal its presence, for in the early stage deposits of tubercle can often be recognised as pale or yellowish nodules, forming minute projections beneath the mucous membrane, the surrounding surface being more or less congested. If the disease has been in progress for some time, one or more small ulcers will be visible, often shallow and irregular in shape, their margins being sometimes sinuous and rounded, sometimes well defined as though punched out. The examination should always be conducted very gently, for in tuberculous disease the bladder does not tolerate any form of instrumentation, consequently a rough use of the cystoscope is very liable to aggravate the cystitis and afterwards render its progress more active.

The conditions liable to be mistaken are *calculus* and *vesical growths.* The absence of a calculus can be proved by sounding the bladder. In tuberculous disease there is no sudden stoppage in the stream of urine as is often observed in cases of stone, and the bladder-symptoms, instead of being relieved by rest in the recumbent position, are often worse at night, the reverse of what usually happens when they are due to the presence of a calculus. In vesical growths, which often occur at a somewhat later period of life, hæmaturia is a more prominent symptom, and is frequently present for a considerable period before evidences of irritability of the bladder appear.

It is often a difficult matter to distinquish *between tuberculosis of the* *bladder and kidney*, especially in the early stages, and it must also be remembered that the two conditions frequently coexist. The presence of pain, tenderness on pressure, and perhaps slight fulness in the lumbar region, would point to implication of the kidney; and a cystoscopic examination of the orifices of the ureters, or their catheterisation in a female, might help to determine the renal origin of the pyuria, which would probably be present under these circumstances.

Treatment.—In the early stage, this should be mainly constitutional, consisting of tonics, cod-liver oil, and a liberal diet; benefit will be derived from a sea-voyage, or residence at the seaside, or in a warm, dry climate. Local applications and irrigation of the bladder are to be avoided, for the introduction of instruments into its interior at this stage often causes irritation and hastens the onset of cystitis. Later on, when cystitis and ulceration are present, relief will often be obtained from washing out the bladder (the fluid being introduced by hydrostatic pressure, if the passage of a catheter into the bladder is still undesirable), from injections of iodoform, or from instillation (*vide* "Treatment of Chronic Cystitis"). In the more advanced stage, or when this treatment fails to give relief and the symptoms are becoming more urgent, suprapubic cystotomy should be performed, and the ulcers either cauterised or curetted. A number of cases have recently been recorded where, as the result of this treatment, the ulcers have cicatrised, the bladder-symptoms subsiding, and the urine also regaining its normal condition; in several instances the improvement had continued several years after the performance of the operation.

Ulceration of the Bladder

This, as already mentioned, may occur in the course of chronic cystitis, and be due to the irritation caused by the presence of calculi, foreign bodies, etc. It may also result from the breaking down of tuberculous deposits, and is often met with in cases where a malignant growth, *e.g.* epithelioma, infiltrates the bladder-wall.

When ulceration has taken place, pain and increased frequency in micturition, with blood in the urine, are usually prominent features, preceded or accompanied by other symptoms special to the particular condition upon which it depends.

"*Simple Solitary Ulcer*" is the term applied by Fenwick to a form of ulcer, usually of small size and single, met with on the trigone or posterior wall of the bladder in young adults of both sexes. The early symptoms are frequency in micturition with intermittent attacks of hæmaturia, and in the case of males an almost constant penile pain is generally a marked feature. The urine remains acid and often clear, not containing pus in any quantity. These symptoms may continue for some time, occasionally for two or three years, or even longer, but sooner or later cystitis is set up, and then the surface of the ulcer often becomes encrusted with a deposit of phosphates, portions of which break off and are either passed with the urine or retained in the bladder, increasing in size and then following definite calculi.

In some cases a second, or "contact," ulcer is formed on the anterior wall of the bladder just above the urethral orifice, owing to the mucous membrane at this spot becoming abraded from contact with the rough, phosphatic surface of the basal ulcer when the viscus empties itself.

Diagnosis.—The condition in the early stage is likely to be mistaken for primary tuberculosis of the bladder and also for calculus. From the former, it can only be distinguished with certainty by means of the cystoscope. In the solitary ulcer, the sheen of the rest of the bladder is perfect. In tubercle, the posterior wall is seen patched with areas of vivid red, and peeling from them are white flakes of necrotic tissue (Fenwick). The absence of any family history or other evidence of tubercle, and the fact that the symptoms are not worse at night, would also be in favour of the presence of a solitary ulcer.

Treatment.—Though benefit may be derived from injections of lactic acid (1 to 3 in 100), or nitrate of silver (1 in 1000 to 1 in 4000), their action is uncertain, and better results are obtained by curetting the ulcers, in females per urethram, in males through a perineal or suprapubic opening, this treatment being followed by their cicatrisation, and often by a permanent cure.

CALCULUS VESICÆ

CALCULUS VESICÆ, OR STONE IN THE BLADDER.—Calculus may occur at any age, but is most frequently met with in children, or in adults over forty years of age, being less common during the middle period of life. It occurs more frequently in males than in females, owing to the shortness of the urethra and its power of dilatation in the latter sex, in consequence of which calculi are more readily discharged with the urine.

ETIOLOGY.—The nuclei of most calculi form in the kidney, passing on into the bladder, where they increase in size by successive deposits from the urine. Less frequently, the nucleus forms in the bladder itself, as the result of immediate precipitation of some of the constituents of the urine; or the nucleus may consist of a small particle of inspissated mucus or coagulated blood, or of some foreign body introduced from without. When originating in the kidney, the nucleus usually consists of uric acid or oxalate of lime, consequently any condition which leads to the excessive formation in the urine of uric acid (lithiasis), or of oxalate of lime (oxaluria), acts as a predisposing cause in the formation of calculi. The fact that calculi are more frequently met with in certain countries, or in certain parts of the same country, would indicate that their formation is influenced to some extent by climate, and probably also by the nature of the drinking water. Thus in England, calculus is very prevalent in the eastern counties, especially in Norfolk, and in foreign countries, among the inhabitants of India, Arabia, and Egypt, those who dwell on the banks of the Nile being especially liable to it.

A calculus, if retained in the bladder, enlarges by deposits from the urine, which, so long as the urine is acid, consist of uric acid or oxalate of lime. If, however, cystitis is set up, and the urine becomes alkaline, phosphates are then deposited upon it. This accounts for the fact that many uric acid or oxalate of lime calculi are coated with phosphates, and also explains why calculi, whose nucleus consists of a foreign body, are usually phosphatic throughout.

Varieties.—The common varieties are those consisting of uric acid, oxalate of lime, and phosphates of lime, ammonia, and magnesia; rarer forms are composed of urate of ammonia, carbonate or phosphate of lime, cystine, and xanthine.

Structure.—A stone may be composed throughout of the same material (simple calculus), or much more frequently, of different elements, often arranged in concentric laminæ (alternating or mixed calculus). A common form is one composed of a nucleus or central portion of uric acid; a body or intermediate portion of oxalate of lime, or alternating layers of this substance and uric acid; and a crust or external layer of phosphates. Sometimes two nuclei are present.

Physical Characters.—Uric acid calculi are generally ovoid in shape, with a finely granular or smooth surface, and are of a fawn or brownish colour. They are of hard consistence, and on section present a laminated appearance. *Oxalate of lime* calculi are more globular, with a rough, tuberculated surface, somewhat resembling a mulberry, hence the term, "Mulberry calculus," often applied to them. They are of a dark brown colour, of very hard consistence and heavier than the other varieties, presenting on section a crystalline or laminated appearance.

Phosphatic calculi consist usually of a mixture of triple phosphate (ammonio-magnesium phosphate), and phosphate of lime. They are often larger than the preceding varieties, and not infrequently somewhat irregular in shape, moulding themselves to pouches or depressions in the bladder. They are of a whitish colour and chalk-like in appearance, sometimes laminated on section, and of softish consistence, being much lighter than the preceding varieties. The nucleus usually consists of uric acid or oxalate of lime, or a foreign body, it being uncommon to meet with a calculus which is phosphatic throughout, except in certain cases of chronic cystitis, where they sometimes form in pouches in the bladder.

Cystine, or *cystic oxide*, calculi contain a large quantity of sulphur, and are of a yellowish-gray colour: they present a peculiar waxy appearance, and sometimes become green after exposure to the air.

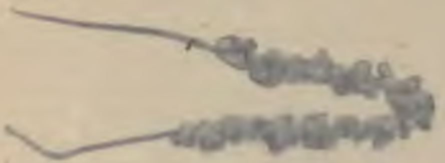
Number.—Calculi are usually single, especially in children, but they may be multiple, and under these circumstances they often present a facetted surface from rubbing against one another.

Size.—Though calculi present considerable variation in size, sometimes attaining considerable dimensions, they are usually about a half to one and a half inches in diameter, as met with in practice. The largest are generally those which are mainly of a phosphatic nature.

Position.—Calculi usually lie loose and movable in the bladder. In adults who are the subjects of enlarged prostate, they often lie in pouches or depressions behind the gland, under these circumstances not changing their position. Occasionally, they are encysted, lying partly or entirely in saccules in the bladder-wall.

Spontaneous fracture of a calculus sometimes occurs in the bladder, the causation of which is difficult to explain. It has been variously attributed to pressure from the contraction of a hypertrophied bladder-wall; to chemical changes in the interior of the stone; to infiltration of urine into a crack or fissure; and in cases where two or more calculi are present, to concussion of one against another.

The SYMPTOMS of stone admit of considerable variation, depending somewhat on its size and shape, and also upon its nature, one which is rough and irregular generally causing more pain than another whose surface is smooth, especially if cystitis is present. They are also modified to some extent by the position of the stone and the age of the patient. *Pain* is usually complained of in the bladder, especially about its neck, and also in surrounding parts, *e.g.* perineum, groin, thigh, and end of the penis. It is generally increased by exercise, or any sudden or jolting movement, and is worse towards the end of, or immediately after, micturition, for, when the bladder empties itself, the stone falls upon, and is pressed against, the neck the most sensitive portion of the viscus. Pain is generally a prominen symptom in children, owing to the fact that at this age there is hardly any base or fundus to the bladder, consequently the stone always tends to res



1

FIG. 1.—Hair-pin coated with [illegible] phosphates, removed from a female [illegible] been present for about six weeks.

2

FIG. 2.—Phosphatic calculus from a female bladder, containing a hair-pin as its nucleus.

3

FIG. 3.—Oxalate of lime calculus, [illegible] surface, coated in places with a layer of phosphates. [illegible] From a male, aged [illegible] years. [illegible]

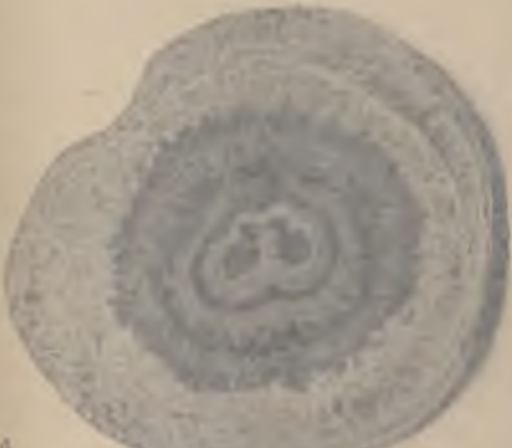


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FIG. 4.—Section of a calculus consisting [illegible] of oxalate of lime. The body consists of [illegible] uric acid and oxalate of lime, and [illegible] extreme layer composed of phosphates. [illegible] (Nat. size.)

[illegible]

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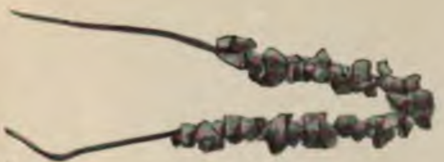

1

FIG. 1.—Hair-pin coated with a deposit of phosphates, removed from a female bladder, where it had been present for about six weeks.

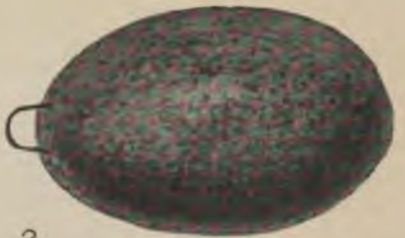

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FIG. 2.—Phosphatic calculus from a female bladder, containing a hair-pin as its nucleus.

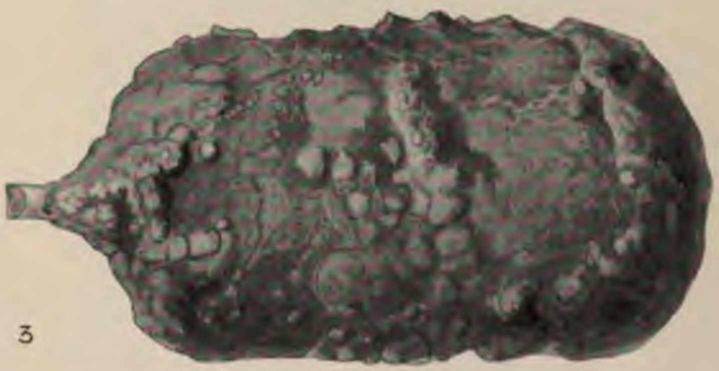

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FIG. 3.—Oxalate of lime calculus, showing a tuberculated surface, coated in places with a layer of phosphates. Projecting from one end of the calculus is a piece of glass rod. From a male, aged 28 years. (Nat. size.)

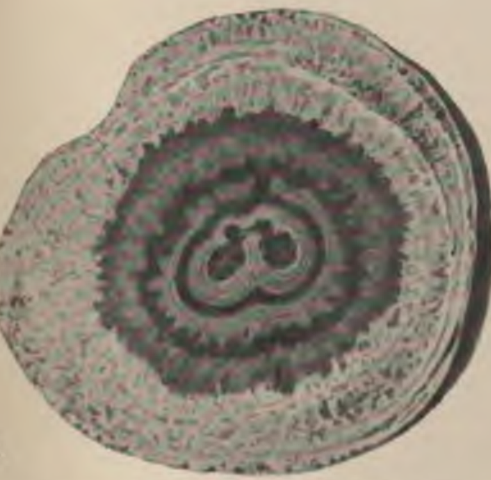

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FIG. 4.—Section of a calculus containing a double nucleus of oxalate of lime. The body consists of alternating layers of uric acid and oxalate of lime, and there is a thick crust or extreme layer composed of phosphates. From a male. (Nat. size.)

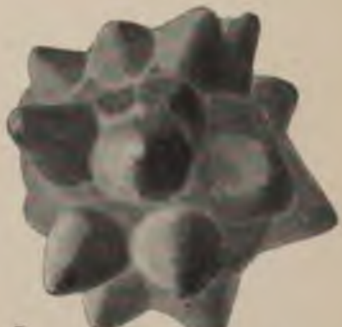

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FIG. 5.—Oxalate of lime calculus with an unusually spiculated surface. From a male, aged 52 years. (Nat. size.)

upon its neck. In adults, when the prostate is enlarged, pain is often less severe and may be altogether absent, for the stone then rests in a depression behind the gland, "post-prostatic pouch," which prevents it from coming into contact with the sensitive neck, even when the bladder is empty. For a similar reason, when encysted, pain may be slight or sometimes absent.

Sudden interruption in the flow of urine is often produced owing to the stone falling upon the neck of the bladder and blocking temporarily the vesical orifice of the urethra, but as in the case of pain, this sign may be absent when the stone is encysted or lies in a pouch behind an enlarged prostate.

Hæmaturia, though not as a rule a prominent symptom, is often present from time to time, especially after any active exercise. It has the usual characteristics of vesical hæmorrhage, the blood in many cases only appearing at the end of micturition, so that the urine first passed is clear, the last few drops being of a bright red or pinkish colour, and sometimes containing small clots.

Frequent Micturition, often accompanied by more or less straining, especially in children, is almost always a prominent symptom, and an altered condition of the urine is usually observed after a time, *pyuria* and other evidences of chronic cystitis being present in consequence of the irritation caused by the calculus. In addition to the preceding, *one or more of the following symptoms* are frequently met with, viz. incontinence or retention of urine, priapism, constant handling of the prepuce in children in order to relieve the pain, which is referred to the end of the penis, the foreskin in consequence becoming elongated, and often presenting an inflamed appearance; irritation of the rectum with straining at stool, causing prolapse in children, and hæmorrhoids in adults; in children a hernia is often produced from the straining efforts in passing water. *The most certain evidence of the presence of a stone is produced by sounding the bladder*, a characteristic ring or click being heard when it is struck with the end of a "sound," the metallic instrument used for this purpose. The bladder should contain a small quantity of fluid, either urine or some antiseptic lotion, and every part of its interior should be carefully explored, the patient lying on his back, with the buttocks slightly raised.

The size of the stone can be estimated by passing a lithotrite and seizing it between the blades of the instrument, for by means of the scale at the other end, the distance between the blades can be accurately measured. Another method of judging, but only approximately, of the size of the stone is by gently drawing the beak of the sound over its surface without moving it. In children, a calculus can often be detected by "bimanual palpation," that is, by introducing a finger into the rectum and pressing with the other hand over the abdomen above the pubes, when the bladder is empty; a good idea of the size of the stone can also be obtained at the same time, especially if the patient is anæsthetised.

The nature of the stone can often be made out by observing the character of the ring elicited on striking it with a sound; if clear and metallic, the stone is of hard consistence, either uric acid or oxalate of lime; if dull, it is of a softer structure, at any rate externally, having a phosphatic encrustation of some thickness. If the surface is rough and tuberculated, the calculus probably consists of oxalate of lime; if smooth, either of uric acid or phosphates. The reaction of the urine will also throw some light upon its nature, uric acid and oxalate of lime calculi forming in acid urine, phosphatic in alkaline urine.

When two or more calculi are present, it will often be found that upon gently rotating the sound, a stone is struck in different directions; or upon seizing one stone in the blades of a lithotrite and moving it in the bladder, it will be felt to strike against another.

An encysted calculus can be recognised by the fact that it is always felt in the same situation, being immovable on alteration of the position of the patient, and also when touched with a sound or seized with a lithotrite. When lying completely within a saccule, not projecting into the bladder, it is usually impossible to detect it with a sound, and in many cases it can only be recognised by means of the cystoscope. Care must be taken not to mistake a roughened bladder-wall, especially when coated with a deposit of phosphates, for an encysted calculus. In cases of doubt, the cystoscope would solve the difficulty.

In the case of children, a dull "thud" is sometimes produced by the sound striking against the sacrum, or some other part of the bony wall of pelvis, but it is quite different from the ring of a calculus.

A small calculus, when difficult to feel with a sound, can often be detected by washing out the bladder with the evacuator employed in lithotrity. The fluid, as it rushes back from the bladder into the evacuator, carries with it the stone, often dislodging it, if it has been lying hidden between folds of mucous membrane, and a distinct ring or click will be heard when it strikes against the end of the catheter.

When the prostate is enlarged and the stone lies in a depression or pouch behind, it is very liable to be overlooked unless this part of the bladder is specially explored with the sound. Pressure upon the base of the bladder by the finger in the rectum, and elevation of the pelvis, will often facilitate its recognition under these circumstances.

Though the presence of calculi in the bladder has recently been demonstrated with success in the case of children by means of the X-rays, this method of diagnosis has not, as yet, been found of any practical value in adults.

DIAGNOSIS.—Vesical calculus has to be distinguished from other affections of the bladder, and also from certain conditions of the kidney, all of which may be accompanied by somewhat similar symptoms, viz. pain and increased frequency in micturition, with the presence of blood in the urine.

The conditions of the kidney most likely to be mistaken are renal calculus and pyelitis, especially the tuberculous variety. In renal calculus there is usually a history of attacks of renal colic, and in both affections some tenderness with increased resistance will probably be experienced on pressure over the region of the kidney; when the seat of tuberculosis, slight fulness of the kidney can often be detected. The vesical symptoms are not as a rule so prominent; if blood is present in the urine, it is uniformly diffused, communicating to it a characteristic smoky tint, and clots are absent.

The affections of the bladder which are liable to be mistaken for stone are chronic cystitis, tuberculous disease, tumours, and enlargement of the prostate. In all cases of chronic cystitis, when no cause is apparent, *e.g.* urethral stricture, foreign bodies, tumour, prostatic disease, etc., the possibility of a stone being present should be borne in mind and the bladder should be carefully sounded. Without this precaution, it is impossible to exclude calculus as the cause of the cystitis, for if accompanied by ulceration of the mucous membrane, pain and increased frequency in micturition will be prominent symptoms, and blood will often be found in the urine, as is the case when a stone is present.

When chronic cystitis is met with in cases of enlarged prostate, and is of a severe nature, not yielding to ordinary treatment, the post-prostatic pouch should be carefully explored with a sound, for, as already mentioned, the presence of a calculus in this situation is often unattended by the usual symptoms of stone. Tuberculous disease of the bladder in the early stages is accompanied by symptoms which may closely simulate those of stone for diagnosis. In most cases of vesical tumours, hæmorrhage is a much more prominent symptom. It is usually more profuse and often the earliest to attract attention, being at the outset unattended by pain or other evidences of vesical irritation, coming on suddenly without any obvious cause, and just as suddenly ceasing. Intermittent attacks of painless hæmaturia are not usually met with in cases of stone. After a time, all the usual evidences of chronic cystitis will appear, and though in many cases the early history of the attacks of bleeding will point to tumour, it is often impossible in the later stages to make a certain diagnosis without a cystoscopic examination, or sounding the bladder.

In children, many of the symptoms of stone may be caused by the irritation of an excess of uric acid in the urine, which under these circumstances occasionally contains traces of blood. On exploring the bladder, a roughened, almost gritty condition of its wall is sometimes felt, "the extreme acidity of the urine exciting a spasmodic contraction of the muscular fasciculi of the bladder and throwing them into ridges or folds against which the point of the sound impinges" (Morgan). The symptoms all subside as soon as the urine has been rendered less acid by means of drugs.

TREATMENT.—A calculus may be removed from the bladder by a crushing operation, *Lithotrity*; or by a cutting operation, *Lithotomy*.

LITHOTRITY.—The stone is crushed as it lies in the bladder by means of a lithotrite, which is introduced per urethram. Formerly, it was the custom to crush the stone at several operations, or "sittings," as they were termed, each lasting only a few minutes, and then to leave the fragments in the bladder to be afterwards passed with the urine by the natural expulsive efforts of the patient. At the present day, the operation is usually completed at a single "sitting," which in the case of a hard stone of some size, may last for a considerable time, *e.g.* for an hour, or even longer; at the same "sitting" all the fragments are removed through a large catheter or "evacuating tube," by the suction action of a special syringe, or "evacuator."

This procedure, which is known as Litholapaxy, was introduced by Bigelow of Boston, and has many advantages over the old-fashioned method of performing the operation, for by removing all the fragments at once, the pain and irritation, which would be caused by their retention in the bladder and their subsequent passage along the urethra, are altogether prevented. In performing Litholapaxy the pelvis should be slightly raised and the bladder should contain a few ounces of some antiseptic fluid: the lithotrite is then introduced, and the stone is seized and crushed, the process being repeated until all the fragments are reduced to small particles. The lithotrite is then withdrawn and an evacuating catheter introduced and connected with the evacuator, which is filled with warm boric lotion. On compressing the bulb of the evacuator, fluid is forced into the bladder; on relaxing the pressure the bulb refills, and the return current from the bladder carries with it the particles of the stone, which fall into a trapped receiver, where they remain. This is continued until all the débris of the stone is removed, viz. until no more fragments come away, and the ringing sound produced when they strike against the end of the catheter in the bladder has ceased. In cases where some fragments still remain in the bladder, being too large to pass through the catheter, it will be necessary to re-introduce the lithotrite and crush them again, as it is very important that every fragment should be removed; for if any are left behind and not subsequently expelled with the urine, they may act as the nuclei of fresh calculi. No special after-treatment is, as a rule, required, beyond keeping the patient in bed for a few days, and if the operation has been a prolonged one, taking the usual precautions to guard against the effects of shock.

The conditions suitable for the performance of lithotrity are as follows:—

1. The stone should be of moderate dimensions; if of a hard nature, consisting of uric acid or oxalate of lime, it should not exceed 1½ inches in diameter; if phosphatic, this size may, however, be considerably exceeded.

2. The stone should be movable and accessible; if encysted, it will often be impossible to seize it with the lithotrite, and it is frequently difficult to do so when it lies in a depression in the bladder-wall, or in a deep post-prostatic pouch behind an enlarged and prominent median lobe. Moderate enlargement of the prostate is no bar to lithotrity.

3. The urethra must be in a healthy state: if a stricture is present, lithotrity is impracticable until the canal is restored to its normal condition.

4. The presence of cystitis does not usually contra-indicate lithotrity, unless the symptoms are of a severe nature. If, however, the urine is foul and alkaline, containing a large quantity of pus, and the bladder is in a highly sensitive state, or contracted with thickened walls, so that it will not retain more than two or three ounces of fluid, then lithotomy often gives better results, for the free after-drainage, which can be maintained so long as the lithotomy wound remains open, always has a beneficial effect upon the inflamed condition of the bladder-wall.

5. In cases where calculus is complicated by renal disease, it is a question, whether, other conditions being favourable, lithotrity or lithotomy should be performed. Though under these circumstances both operations are attended by a somewhat high rate of mortality, the general opinion appears to be that lithotrity involves less risk to life.

Perineal Lithortity.—The old operation of perineal lithotrity has recently been revived and advocated by Reginald Harrison, in cases where a stone of large size is complicated with a pouched bladder or enlargement of the prostate gland. As the term implies, the bladder is entered through a perineal incision made into the membranous portion of the urethra. The prostate and neck of the bladder are dilated instead of being incised, and the calculus having been crushed by a special lithotrite of great strength, introduced through the perineal wound, the fragments are then removed by the same route.

Lithotrity in Male Children.—Until a few years ago, it was believed that lithotrity was not a safe or an easy operation in male children on account of the small size of the bladder and urethra in young subjects, which rendered it difficult to introduce and manipulate instruments of sufficient strength to crush a stone. The delicate but powerful lithotrites, which are specially made for children, have overcome this difficulty, and calculi of moderate size, *e.g.* ¾ inch or even an inch in diameter, can be crushed as readily and as successfully in young subjects as in adults, provided that all the fragments are removed at the time of operation. The urethra of a boy three or four years of age will usually admit of the introduction of a catheter No. 7 or 8 (Eng. scale), and the operation has been successfully performed in children as young as 18 months.

Lithotrity in Females.—A crushing operation is not often required in females, owing to the power of dilatation of the urethra, but it gives good results in cases where a stone is too large to be removed intact by this route.

LITHOTOMY.—Owing to the improvements in lithotrity during recent years, lithotomy is now performed much less frequently than was previously the case, a cutting operation being required only under certain special conditions, the most common of which are:—(1) A stone of large size and of extreme hardness; (2) an encysted condition of a stone; (3) A combination of stone, and marked enlargement of the prostate gland, where the calculus lies in a deep post-prostatic pouch. When one or other of these conditions is present, lithotomy is generally indicated, and the surgeon has then to choose between the recently revived suprapubic operation, and what we are now beginning to regard as the "old-fashioned" perineal or lateral operation. Of the two routes to the bladder, the suprapubic presents many advantages over the perineal, for it affords special facilities for the extraction of large or encysted calculi. In cases of stone associated with prostatic hypertrophy, the enlarged gland is left untouched, and therefore free from the bruising and laceration which necessarily accompany the lateral operation under similar conditions. It also does away with all risk of hæmorrhage from the prostatic plexus of veins, often a somewhat troublesome complication of lateral lithotomy when performed in persons advanced in years. In cases of encysted calculi, by permitting the whole of the interior of the bladder to be inspected, it renders their detection, as well as their removal, much easier. It also prevents any risk of injury to the rectum, the ejaculatory ducts, and the artery to the bulb, the division of the latter in lateral lithotomy being sometimes attended by troublesome hæmorrhage. Consequently, perineal lithotomy, whether by a lateral or a median incision, is becoming performed less frequently year by year, the suprapubic operation gradually replacing it.

In *suprapubic lithotomy* the bladder is entered by an opening made through the abdominal walls in the middle line immediately above the pubes, care being taken to avoid wounding the anterior reflection of the peritoneum. When the bladder is empty this fold reaches down to the pubes, but when it is moderately distended (*e.g.* containing 6-10 oz. of fluid), the fold is raised and a suprapubic interval is left uncovered by peritoneum, through which the viscus can be safely entered. Before opening the bladder, it should be secured by two silk ligatures passed through its walls on either side of the median line, otherwise it often happens that immediately it is incised, the fluid contents escape, and the viscus, as it empties itself, sinks back into the pelvis, so that it may be difficult to find the opening again, especially in the case of a stout subject. The bladder having been opened in the middle line between the two ligatures, care being taken to avoid wounding the peritoneum and any overlying veins, its interior is explored with the finger in order to ascertain the position of the stone, and also whether more than one is present. The stone or stones are then extracted by means of forceps or with a scoop, and all bleeding is arrested. As regards the after-treatment of the wound, this varies, depending upon the condition of the bladder-wall and the urine. If both are healthy, the opening in the bladder may be closed by sutures, which should include only the muscular coat, and in many cases it will heal by primary union. It is usually advisable to leave the wound in the abdominal wall partly open, so that if the sutures give way and there should subsequently be an escape of urine, there may be an outlet for it. It is also advisable to tie in a catheter per urethram, or to draw off the urine at frequent intervals for some days, to prevent any distension of the bladder taking place. If, however, the urine is in an unhealthy condition, and the viscus presents well-marked signs of chronic cystitis, the wound in its walls should be left open, a drainage-tube being inserted for two or three days, healing under these circumstances taking place by granulation. To correct the toxic state of the urine, the bladder should be washed out regularly with some antiseptic lotion, *e.g.* boric acid, and powdered boric acid should be frequently dusted into the suprapubic opening. By these means the unhealthy condition of the urine is counteracted, and the cystitis is relieved; at the same time, the risk of pelvic cellulitis, which in former years was a frequent and often a fatal complication of the operation, owing to infiltration of toxic urine into the cellular tissue in the neighbourhood of the suprapubic wound, is almost entirely prevented. The wound gradually contracts, and usually closes in the course of three or four weeks, and the free drainage which is maintained so long as it remains open, always has a beneficial effect upon the cystitis.

Lithotomy in females is rarely required, most calculi being capable of removal per urethram, especially if first reduced in size by a lithotrite. If, however, too large or too hard to be extracted by this route, then suprapubic lithotomy should, as a rule, be performed in preference to vaginal lithotomy, *i.e.* by means of an incision through the anterior vaginal wall.

Lithotomy in children.—In cases where lithotrity is contra-indicated by the size of the stone or other conditions present, suprapubic lithotomy should be performed, for the bladder, being an abdominal rather than a pelvic organ in young subjects, is readily accessible by this route. The suprapubic operation is not only extremely successful in male children, but it also obviates all risks of sexual impotence in after life, an occasional sequel of lateral lithotomy in boys, from injury to the seminal vesicles or their ducts.

Recurrence after operation.—It is only in a small proportion of cases that calculi recur after removal. When recurrence takes place after lithotomy, it is probably due to the same constitutional causes which led to the formation of the calculus in the first instance, another forming in the kidney and passing on into the bladder, and it is most likely to occur in cases where more than one calculus was found in the bladder at the time of operation. When following lithotrity, it may be due to the same cause, or more probably because the bladder was not completely cleared of all the fragments, one of these remaining behind and acting as the nucleus of a fresh calculus. That the latter view is correct is proved by the fact that recurrence is more common after lithotrity than after lithotomy, and it is met with much less frequently now than in former years, in consequence of the improved means at our disposal for removing all the fragments at the time of operation.

The *treatment* of secondary calculi is identical with that already described, but in performing suprapubic lithotomy for the second time on the same patient, it will often be found that the anterior reflection of peritoneum is drawn down and fixed close to the upper border of the pubes, in consequence of the cicatricial contraction of the parts in the neighbourhood of the old wound, so that unless care is taken, it is liable to be injured in exposing the anterior surface of the bladder.

TUMOURS

The bladder is frequently the seat of new growths, which may be either primary or secondary, the former originating in its walls, the latter involving it secondarily, usually by direct extension from some adjacent part. The *primary* growths usually met with are Papilloma, Sarcoma, and Carcinoma: much rarer varieties are Adenoma, Myoma, Myxoma, and Angioma: instances of Osteoma and Enchondroma have been recorded. As regards the relative frequency of the three most common forms of growth, sarcoma is much less rare than was formerly believed, many instances of so-called "villous cancer" being really of this nature. In 22 consecutive cases of vesical growth examined microscopically by the writer, their structure was as follows:—papilloma, 9; sarcoma, 7; carcinoma, 6 (epithelioma, 4; encephaloid, 1; scirrhus, 1). Primary tumours are much more common in males than females, the urinary and genital disorders of the former sex, by acting as local causes of irritation, probably predisposing to their formation. *Secondary* tumours are usually of a cancerous nature, and due to direct extension from the uterus or vagina in females, and from the rectum and prostate in males. Dermoid and hydatid cysts may also involve the bladder, usually secondarily, very rarely as primary affections.

PAPILLOMA OF BLADDER.—Papilloma usually presents itself as a soft flocculent growth made up of floating tufts or feathery processes, not unlike the villi of the chorion, hence the term "villous tumour" often applied to it. Growths of this nature may be single or multiple, and pedunculated or sessile; they spring from the mucous membrane or submucous tissue, not involving the deeper coats, their favourite situation being the trigone and the neighbourhood of the ureter. In some cases there is no distinct tumour, the affected portion of the bladder-wall presenting a velvety or a shaggy flocculent appearance, occasionally for a considerable extent. The processes, which may subdivide several times and are often of some length, are composed of a delicate stroma of connective tissue, covered with a basement-membrane and several layers of epithelial cells, and running in the stroma are numerous fine capillary vessels. The sessile growths (fibro-papilloma) are usually of firmer consistence than the pedunculated, especially towards their bases, containing a denser fibrous stroma, mixed with which there is often a quantity of unstriped muscular fibre. They present considerable variation in size, *e.g.* from a pea to a small orange, or even larger, and when pedunculated the pedicle may measure an inch or more in length. Though papillomata are essentially innocent tumours, often remaining so for many years, yet after a time, just as happens with similar growths in other parts of the body, they may change their nature, especially towards their base, and take on malignant action. Undergoing either cancerous or sarcomatous degeneration, they then infiltrate the bladder-wall deeply, and growing rapidly, soon attain a large size, often completely filling the interior of the viscus. Papillomata are rarely met with before puberty, but are not uncommon at any age between 30 and 70 years.

SYMPTOMS.—In the early stage, attacks of *painless hæmaturia*, with intervals of complete freedom from bleeding, are usually the only symptoms present. The urine is generally of a pinkish or bright red colour, and often contains distinct clots. The blood is usually present in greater quantities at the end than at the commencement of micturition, and at the very termination of the act almost pure blood may be passed. In the early stage the attacks of bleeding may last for a day or for several days, coming on without any warning or apparent cause, and then just as suddenly ceasing, but occurring again after a variable interval, often of some months. This condition of *intermittent hæmaturia* may sometimes continue for years, but as time goes on the attacks become more frequent, recurring at shorter intervals and lasting for a longer period on each occasion, until at length blood is never absent from the urine. As the hæmaturia is at first unaccompanied by pain or other symptoms, it will often be found that the patient has paid little attention to it. Sooner or later, however, as the growth increases in size, and especially if it involves or is situated in the neighbourhood of the neck of the bladder, acting like a foreign body, it becomes a source of irritation and sets up *chronic cystitis*. All the symptoms of cystitis then appear, micturition becoming painful and frequent and pus appearing in the urine, which presents the usual evidence of the existence of this condition.

Other Symptoms which may develop are as follows:—Difficulty in micturition, owing to the growth blocking the vesical orifice of the urethra, most likely to occur when it is situated near the neck of the bladder, or when it is pedunculated. Retention of urine, due to the same cause, or to impaction of clots of blood in the urethra. Sudden stoppage in the flow of urine, due to either of the causes last mentioned. Protrusion of the growth from the external orifice of the urethra in females. Expulsion of portions of the growth in the urine, in the form of villous processes which have become detached from its surface; these appear as fine shreds of tissue, which, examined microscopically, show the characteristic structure of the villi, viz. a central axis containing one or more capillary vessels, and invested with several layers of epithelial cells.

Owing to their soft structure it is usually impossible to detect a papilloma on sounding the bladder, on digital examination per rectum or vaginam, or upon bimanual palpation. *On examination with the cystoscope* the villous processes can be seen floating and moving to and fro in the fluid contents of the bladder; they are usually of a pinkish or a rose-red colour, and their small vessels are sometimes distinctly visible. By this means not only can the presence of a growth be verified, but in many cases its situation, as well as its size and the nature of its connection, can be accurately estimated. It will often be found that after a cystoscopic examination or sounding the bladder, bleeding is excited or increased, and when copious, this is of itself always suspicious of the presence of a growth. If a hollow sound is used, or if the bladder is washed out through a catheter, it will often happen that on withdrawing the instrument it will bring away a small portion of the growth in its eye. In the case of females a digital examination of the bladder through the dilated urethra will always reveal the presence of a growth. In cases of papilloma, just as in other forms of vesical tumour, *secondary changes of a degenerative nature are sooner or later set up in the kidneys*, in consequence of the back-pressure which is produced, when the growth offers any obstruction to the entrance of the urine into, or to its escape from, the bladder. The conditions most commonly met with are interstitial nephritis and hydronephrosis; when cystitis is present, septic inflammation may spread upwards along the ureters to the kidneys, setting up pyelitis, often accompanied by suppuration in the substance of the gland. The prognosis in papillomata is, therefore, very unfavourable, for if allowed to run their course, which may last for years, as a rule they ultimately prove fatal. Though death may be due to mere loss of blood, a condition of profound anæmia being produced, and the patient's strength and vital power being reduced by this cause, as well as by the severe suffering due to the supervention of cystitis, yet there is generally superadded one or other of the renal complications just mentioned. In cases where the growth takes on a malignant action, the disease then runs a more rapid course, soon terminating fatally.

SARCOMA OF BLADDER.—Sarcoma is frequently met with in the bladder, and it is probable that many of the cases which were formerly described as "villous cancer" were really of this nature. It usually occurs as a primary growth, consisting of round, or of a mixture of round and spindle cells: examples of lympho- and myxo-sarcoma, and also of the alveolar variety, are occasionally met with. It springs from the submucous tissue, but soon tends to invade the other coats, so that after a time the whole thickness of the bladder-wall may become involved in the disease, which occasionally infiltrates the surrounding parts. Sarcoma is met with in children as well as in adults, and though vesical growths are not common in young subjects, the majority will be found to be of this nature. When occurring in adults it often forms a prominent mass of considerable size, occasionally pedunculated, but much more frequently sessile, and usually springing from the base or posterior wall. The growth may be firm and fleshy throughout, and irregularly lobulated or fissured, presenting a cauliflower-like appearance. In other cases the surface is soft and villous, the deeper portion being of firm consistence, or the growth may be soft throughout, so closely resembling a papilloma that, without a microscopic examination, it is impossible to make out its true nature. When occurring in children, sarcoma frequently appears in the form of multiple polypoid growths, varying in size from a pea to a walnut, often forming large masses (resembling a bunch of grapes), and more or less completely filling the bladder. The surface of the growths under these circumstances is generally smooth and devoid of villi. Tumours of this nature, which are usually examples of myxo- or fibro-sarcoma, were formerly described as "mucoid or fibroid polypi" of the bladder.

SYMPTOMS.—*Though the general symptoms are of a similar nature to those of papilloma, the disease as a rule runs a more rapid course,* for the attacks of hæmaturia soon become more frequent and more profuse, and evidences of chronic cystitis and secondary renal disease more rapidly develop. Thus, while a case of papilloma may last for many years, *e.g.* five, ten, or even fifteen years, it will be found that in sarcoma, if allowed to run its course, a fatal result usually takes place in from one to three or four years. As the disease advances, physical signs of its presence can often be detected, for if of firmish consistence, owing to its tendency to infiltrate the bladder-wall, it can often be felt on examination per rectum or per vaginam, or upon bimanual examination; upon exploring the bladder with a sound an obstruction is sometimes encountered, or the movement of the instrument in the bladder can be felt to be limited. In addition to villous processes, distinct portions of growth are occasionally passed in the urine, sometimes large enough to block the urethra and cause a temporary stoppage in the stream. On examination with the cystoscope, the floating processes can be seen when the surface is villous: more frequently the appearance presented is that of an irregular or lobulated surface of a grayish or brownish colour, ulcerated in places, with tags of adherent necrotic tissue which may be distinguished from villi by their paler colour and absence of any signs of vascularity.

When sarcoma occurs in children in the form of the polypoid growths referred to, the most prominent symptom is generally difficulty in micturition, followed after a time by retention or incontinence of urine, hæmaturia being frequently absent or only present to a slight extent. This is explained by the less vascular nature of the growths and by the absence of a villous surface. Cystitis is soon set up, and in other respects they run the same course as in adults, soon terminating fatally. Secondary deposits are only very rarely met with in the adjacent lymphatic glands, but may occur in internal organs and other parts of the body, as is the case in sarcoma generally.

CARCINOMA OF BLADDER.—CARCINOMA may attack the bladder as a primary or secondary affection in the form of (1) glandular or acinous cancer; (2) or epithelioma.

1. *Glandular or acinous carcinoma* is usually of the encephaloid variety, scirrhus being extremely rare. *Encephaloid* cancer generally occurs as a primary growth, and in its physical characters it closely resembles sarcoma. *Scirrhus,* which has only been met with in a few instances, manifests itself as an infiltration or thickening of the bladder-wall, usually without forming a distinct tumour, and under these circumstances the viscus presents a contracted appearance, its power of expansion being interfered with in consequence of the induration of its walls. The mucous membrane may for a long time remain free from ulceration, showing little change beyond corrugation of its surface.

2. *Epithelioma,* when affecting the bladder primarily, usually appears as a squamous-celled growth. Commencing in most cases at the base, it rapidly spreads, infiltrating the entire thickness of the bladder-wall, which presents a ragged and irregular raised surface, ulcerating in places, and often coated over with a deposit of phosphates. The bladder-wall is thickened, and in the neighbourhood of the patches of ulceration, the margins of which are raised and indurated, there are often numerous small hard nodules which after a time also break down and ulcerate. Sometimes two or more epitheliomatous growths separated by healthy tissue are present in the same bladder, and under these circumstances it has been suggested that one patch becomes inoculated from another, owing to contact when the viscus is empty, hence the term "contact epithelioma," sometimes applied to this condition. Secondary epithelioma is more frequent in females than in males, being more commonly due to extension from the uterus or vagina than from the rectum: in the former case it may be either a

columnar or squamous-celled growth ; in the latter it is usually columnar. In carcinoma of the bladder, secondary deposits are occasionally found in the pelvic and lumbar lymphatic glands when the disease has been in existence for some time, and under these circumstances infiltration of the pelvic cellular tissue may also occur, in some cases giving rise to a swelling which is visible externally. Occasionally perforation of the bladder-wall takes place, followed by extravasation of urine, or by the formation of a vesical fistula.

Symptoms.—In encephaloid cancer the symptoms and course of the disease are so similar to those of sarcoma, that it is often impossible to distinguish between them except by examination of a portion of the growth. In epithelioma the early symptoms are not, as a rule, intermittent attacks of hæmaturia, but rather *those indicative of irritation of the bladder*, cystitis developing at an earlier stage, probably in consequence of the infiltrating nature of the growth; the result is that from the outset pain and frequent micturition are usually present. Sooner or later blood appears in the urine, varying in amount, the hæmorrhage being irregular in its occurrence, and depending to a large extent upon the presence or absence of ulceration of the surface of the growth. The thickened condition of the bladder-wall, when the base is involved, can be felt on examination per rectum or per vaginam, and on exploration with a sound the rough, hard surface of the growth can often be distinguished. On cystoscopic examination the appearance presented in encephaloid, and often in epithelioma, is very similar to that met with in sarcoma. In many cases of epithelioma there is, however, more distinct evidence of ulceration, the raised margins and brownish irregular base, with adherent shreds of grayish necrotic tissue, being plainly visible, and contrasting vividly with the smooth pinkish surface of the adjacent healthy mucous membrane. In carcinoma, secondary renal changes are more rapidly produced, for in consequence of the infiltrating nature of the growth the orifices of one or both ureters are likely to be soon obstructed, while from the early supervention of cystitis and its tendency to spread upwards to the kidneys, septic complications often develop in these organs at an earlier stage than in the course of other forms of bladder tumour.

Diagnosis.—In cases of hæmaturia of vesical origin, especially when of a painless nature and recurring in attacks separated by intervals of complete freedom, the presence of a growth should be suspected. When this condition has existed for some months, or for a year or more, and when the attacks tend to increase in frequency and also in severity, but still remain unattended by any evidence of vesical irritation, the growth is probably a papilloma, less commonly of a sarcomatous nature. In cases where the bleeding is irregular, or more or less persistent, but preceded or accompanied from the first by pain and increased frequency in micturition, the growth is more likely to be cancerous, viz. epithelioma. Other causes of vesical hæmorrhage must also be considered, *e.g.* stone, tuberculous ulceration, solitary ulcer, enlargement of the prostate, and cystitis. In prostatic hæmorrhage, except when produced by instrumentation, the blood is usually venous in origin and therefore of a darkish colour, not pink or bright red, as is usually the case when a growth is present, and there will be other signs pointing to the prostate as its source. In hæmorrhagic cystitis the bleeding is sometimes so free that it is at first attributed to a growth, but there will be no history of previous or recurrent attacks, and the patient will present other evidences of acute cystitis. The presence of a tumour can be verified by a cystoscopic examination, and in many cases by the detection of particles of the growth in the urine, which should always be carefully examined in suspected cases. When of a cancerous nature it can often be felt on examination per rectum or per vaginam.

Treatment.—The *palliative* treatment consists in the adoption of measures to arrest hæmorrhage and to relieve the symptoms due to cystitis when this condition has supervened. To check bleeding, hæmostatics should be given internally, and those which are most useful are ergot, hamamelis, turpentine, and gallic acid. Though the value of the last-named drug as an internal astringent has recently been questioned, the writer has seen marked benefit follow its administration (given in 10 gr. doses frequently repeated) in many cases of vesical hæmorrhage. Local injections are not, as a rule, to be recommended, though irrigation of the bladder with sterilised water or boric lotion (at a temperature of 110°-120° F.) sometimes causes a temporary arrest of bleeding. In some cases the same result may be obtained by injections of hazeline or tinct. hamamelis (ʒij. to ℥j.) undiluted, after the bladder has been washed out with warm boric lotion and then emptied of its contents. The application of an ice-bag to the perineum or over the pubes, and the introduction of ice into the rectum, may also act in a similar manner, the patient being meanwhile kept absolutely at rest in the recumbent position. To relieve the pain and irritable condition of the bladder when cystitis is present, the treatment described for that condition should also be carried out. This form of treatment may often be adopted with benefit, either as a temporary measure preliminary to operation, or, in cases of malignant growth, where on account of the extent and nature of the disease its removal is clearly impracticable.

The *operative* treatment may be considered under two heads, viz. removal of the growth and bladder-drainage.

Removal of a vesical tumour is best effected through a suprapubic opening, as this route affords better access to the bladder than the perineal. In cases of papilloma, when pedunculated, and also in those exceptional cases of sarcoma, where there is a distinct pedicle, the growth can be completely removed, and under these circumstances the cure will often be permanent, but in the sessile variety of papilloma, however thorough the removal, there is always a great tendency for recurrence to take place. In the malignant forms of tumour, viz. carcinoma and most cases of sarcoma, operative interference is not attended by satisfactory results, for unless undertaken in the very early stage, and before they have commenced to infiltrate widely or deeply the bladder-wall, the probability of a permanent cure is very slight, though life may be prolonged by the operation. Excision of a considerable portion of the bladder-wall together with the growth has been performed under these circumstances, the ureters being inserted into the rectum or vagina with resection of the pubes, or symphyseotomy, in order to allow of better access, but, as might be expected, the results have not been sufficiently encouraging to recommend the adoption of this procedure.

In removing a vesical tumour, the bladder should be opened from above the pubes with the same precaution as in suprapubic lithotomy, and the extent and connection of the growth should be carefully ascertained, Fenwick's "caisson-speculum" often proving extremely useful for this purpose. When pedunculated, the pedicle may be twisted off with forceps or divided with scissors; when sessile, the growth should be scraped away with the finger-nail or a curette. It is very important to remove all the base as thoroughly as possible, either dissecting it out with scissors, and then, if possible, closing the wound on the inner surface of the bladder-wall with sutures, or applying the thermo-cautery. The after-treatment of the wound is the same as in the case of suprapubic lithotomy, some surgeons closing the opening in the bladder-wall by means of sutures.

Bladder-drainage, which is best effected through a suprapubic opening, often gives great relief in cases of malignant tumours, where their removal is obviously impracticable, or when it is found to be so after opening and exploring the interior of the bladder. It is indicated in cases where, in addition to free hæmorrhage, cystitis is a prominent symptom, causing pain and dysuria, which cannot be relieved by the ordinary measures. By establishing a permanent outlet, and allowing the urine to continually drain away, the bladder is left in a state of complete rest; the result is, that the bleeding either ceases or is greatly diminished, while the pain and straining which accompany the frequent, and often ineffectual attempts to pass water are at once relieved. At the same time, the cystitis and the septic condition of the urine, usually present under these circumstances, can be corrected by irrigating the bladder through the suprapubic opening. As a palliative measure, bladder-drainage, therefore, affords great relief, not only prolonging life by checking the loss of blood, but also preventing the drain upon the patient's strength by alleviating the suffering caused by the presence of the growth and the cystitis which it excites.

IN FEMALES, vesical tumours, when of small size, and especially if pedunculated, can often be removed through the dilated urethra, it being sometimes possible to draw them down to the orifice of the canal and bring into view their point of attachment to the bladder-wall, from which they can then be dissected or twisted off. If, however, too large or not readily accessible by this route, they should be removed through a suprapubic opening, this method being—in the writer's opinion—preferable to vaginal cystotomy, where an incision is made into the base of the bladder through the anterior vaginal wall.

FISTULÆ

VESICAL FISTULÆ.—Urinary fistulæ connected with the bladder may be divided into two classes, viz., Vesico-abdominal and Vesico-visceral.

1. *Vesico-abdominal.*—In this form the fistula opens externally on the abdominal wall. It may be due to penetrating wounds involving the bladder, or to suppuration in its neighbourhood, as in cases where an abscess bursts internally into the viscus and also externally in the groin or lower part of the abdominal wall. It is also occasionally met with as the result of non-closure of the wound after suprapubic cystotomy. (*Vide* Urachal fistulæ.)

2. *Vesico-visceral.*—In this form there is a communication between the bladder and some viscus or cavity, *e.g.* the intestine or vagina.

(A) *Vesico-intestinal.*—Communications between the bladder and rectum, "Recto-vesical" fistulæ, are not very uncommon as the result of ulceration of the bladder, which may be either of a simple or malignant nature. They may also be due to injury, *e.g.* penetrating wounds involving the rectum and bladder, and occasionally follow certain operations, *e.g.* puncture of the bladder per rectum, or perineal lithotomy, when the rectum is accidentally wounded. Communications with the small or other parts of the large intestine, though less common, are sometimes met with as the result of ulceration, which may be either simple or malignant.

(B) *Vesico-vaginal.*—Communications between the bladder and vagina may be due to similar causes, or the result of sloughing after difficult labour.

SYMPTOMS.—In vesico-abdominal fistula there is a discharge of urine, often mixed with pus, from the external opening, which generally causes excoriation of the surrounding skin. In vesico-intestinal fistula, fæcal matter often passes into the bladder, setting up cystitis, and in some cases gas also makes its way into the viscus from the bowel, bubbles of air as well as fæces being then expelled with the urine ("pneumaturia").

The TREATMENT in these cases must be conducted on general principles. If there is any cause of obstruction to the escape of the urine per urethram, it should if possible be removed. In recto-vesical fistulæ due to malignant disease, whether originating in the bladder or rectum, colotomy will often afford relief by preventing the passage of fæces into the bladder. When due to simple ulceration, the same operation by leaving the bladder free from irritation will often enable the fistula to heal, and when this has taken place the artificial anus may subsequently be closed by a plastic operation. In vesico-abdominal fistulæ of a non-malignant nature, scraping or refreshing the margins of the track, and afterwards keeping the bladder empty by retaining a catheter or frequently drawing off the urine, will often be followed by healing.

The Female Bladder.

The previous description of morbid conditions of the bladder holds good for male and female bladders alike, but owing to the anatomical differences and their associated clinical features, a short article indicating their leading points is here given.

ANATOMY AND PHYSIOLOGY.—The urethra, which is only 1⅜ inch in length and very dilatable, is closely incorporated with the lower portion of the anterior vaginal wall, while the posterior wall of the bladder is separated from the upper portion of the vagina, the cervix and the lower portion of the uterine body by loose connective tissue which permits of free movement of one viscus upon the other. The anterior surface of the upper part of the uterus rests upon the upper part of the bladder. The peritoneum, firmly attached over the fundus of each organ, dips down between them in the utero-vesical pouch, where it is loosely attached. When empty, the bladder lies behind the pubic symphysis, more or less to one side. Its outline, in sagittal section, is oval when contracted, Y-shaped when relaxed. As the bladder fills, it pushes the uterus backward and obliterates, more or less, the utero-vesical pouch. Being relatively broad at the base, it does not rise above the pelvic brim until it contains considerably over ℥xij. of fluid. Early in labour the bladder is pulled up above the brim and remains an abdominal organ until parturition is completed.

PATHOLOGY.—The shortness of the female urethra accounts for the ease with which the bladder becomes infected, and for the facility with which foreign bodies are introduced. Calculi are found less frequently in the female than in the male bladder, because they escape when still small by the short wide female urethra; sometimes, however, becoming impacted at or near the meatus. Owing to its relation to the vagina, the bladder is said to be injured occasionally in coitus. Substances from the neighbouring reproductive organs may enter the bladder, *e.g.* pus from pelvic abscess, bones from ectopic gestation, pessaries from the vagina. Contraction of the bladder during micturition often causes pain by pulling upon inflamed pelvic structures, while a full bladder causes discomfort by pressing on them. Pelvic tumours mechanically obstruct the movements of the bladder, causing irritation and frequency of micturition. By occupying the pelvis, they cause the bladder to rise when full into the abdomen, when they frequently cause retention by compressing the urethra against the symphysis. For example, in normal pregnancy frequency of micturition is common, while in retroversion of the gravid uterus the bladder is lifted above the pubes and retention frequently results from pressure of the cervix upon the urethra. Malignant disease of the reproductive organs rapidly invades the bladder, and is the most frequent cause of vesico-vaginal fistula. Prolonged pressure of the head against the symphysis during labour may cause sloughing of the tissues, resulting in fistula. The early use of the forceps has greatly lessened the frequency of fistulæ of this kind, which are now but seldom seen. Lacerations of the bladder produced directly by obstetric instruments are still less common. When the pelvic floor fails in supporting the viscera which rest upon it, there is downward displacement of its anterior portion, and the anterior vaginal wall, with the urethra and the lower part of the bladder, bulges out at the vaginal orifice. Irritability and frequent micturition are the first symptoms of this downward displacement of the bladder, which is accompanied by retroversion and downward displacement of the uterus. This condition of "sacro-pubic hernia," more generally known as *prolapsus uteri*, must be distinguished from the much rarer condition, true *cystocele*, usually seen in old people, in which the lower part of the bladder, covered by the vaginal wall, bulges out at the vaginal orifice, the uterus and the upper portion of the bladder remaining in their normal position.

Examination—(a) *Bimanual.*—With two fingers in the vagina and the other hand on the abdomen, vesical calculi and foreign bodies can be discovered unless very small; tenderness is recognised and inflammatory thickening of the bladder-wall may be estimated.

(b) *Sounding and Catheterisation.*—When examining the interior of the bladder with a metal sound or a catheter, the instrument should be introduced by the aid of vision into the meatus, when a finger in the vagina guides it into the bladder. If, with the bladder above the brim, the progress of the instrument is arrested by any body (*e.g.* the fœtal head) pressing against the symphysis, the genu-pectoral position should be adopted and a silver male instrument should be used.

(c) *Palpation.*—Cocaine is applied within the urethra, or a general anæsthetic is given. Then, using the lithotomy posture for choice, and with strict aseptic precautions, the urethra is gradually dilated by inserting the little finger, or by passing a series of Hegar's dilators. The index-finger is then passed into the bladder, and, with the second finger in the vagina and the other hand on the abdomen, the whole bladder can be palpated within and without. There is danger of causing permanent incontinence if the urethra be dilated beyond certain limits, namely, 1 in. in diameter in women; $\frac{3}{4}$ in. in young women; and $\frac{11}{16}$ in. in girls.

(d) *Inspection.*—A small tubular speculum is passed through the dilated urethra, the lithotomy position being used. When the hips of the patient are raised on cushions 12 or 16 inches above the level of her shoulders, air rushes into the bladder and balloons it. Reflected light is thrown in, and by moving the speculum the posterior and lower portion of the bladder can be inspected, the openings of the ureters being easily seen. In order to obtain a view of the anterior and upper portions of the bladder it is necessary to employ the electric cystoscope (*vide* Cystoscope).

(e) *Catheterisation of the Ureters.*—When the finger has been passed into the bladder, the vesical openings of the ureters can be felt about an inch from the neck of the bladder, the ureteric ligament extending between them. A fine catheter can be passed beside the finger and directed into either ureter. When this has been done, the urine from the catheterised ureter can be collected in one vessel, while, with a second catheter, the urine from the other ureter can be drawn from the bladder for comparison. Another and easier method is to pass the ureteric catheter into the bladder through a tubular speculum, and, using reflected light, to insert it into the ureter by inspection.

Treatment.—The anatomical relations of the female bladder permit of certain methods of surgical treatment not available in the male. Thus, in cystitis, dilatation of the urethra permits of drainage for a time, which can be indefinitely prolonged by the use of a self-retaining catheter, with which a urinal can be worn. If more complete rest for the bladder be desirable, a vesico-vaginal fistula may be made by cutting through the anterior vaginal wall, with knife or cautery, on to an instrument passed into the bladder *per urethram.* When the cystitis has disappeared, the artificial fistula can be closed by a plastic operation. Again, small calculi and foreign bodies are easily removed through the dilated urethra, stones of larger size demanding preliminary crushing. Still larger calculi and some neoplasms are easily removed by vaginal cystotomy. The wound may be closed at once if the bladder is healthy, or may be left open for a time by way of drainage, and closed subsequently. Stones of the largest size and extensive neoplasms are best dealt with by suprapubic cystotomy. Where the dilated urethra does not suffice it may be stated, as a general rule, that surgeons will prefer the suprapubic route to the interior of the bladder, whether for drainage or for the removal of calculi, foreign bodies, and neoplasms. Gynæcologists, on the other hand, will prefer to reach the bladder through an incision in the anterior vaginal wall.

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Blenorrhœa. *See* Gonorrhea.

Blepharitis. *See* Eyelids.

The Blind, Training and Education of.—The blind as a class are cheerful, happy, and contented, eager for information, and interested in all that goes on about them. Nevertheless, the loss of sight cannot but be regarded as one of the greatest afflictions. The man who has lost his sight has suffered irreparable loss; he who was born blind is shut out from even the imagination of what sight may be. The education of the other senses of the blind, especially of touch and hearing, can be carried to a wonderful degree of perfection, but at the best these are but narrow gateways of knowledge.

The Prevention of Blindness.—During the last fifty years the proportion of blind in the community has been slowly but steadily declining. This may be attributed to various causes, including all those, hygienic and other, which have improved the home-life of the masses; precautions at work; better education, leading those suffering from any disease or injury of the eye to seek skilled assistance without delay; and the great decrease of such diseases as smallpox which formerly accounted for many cases of blindness, and still does so in some countries, as has recently been shown by Solowin, who investigated the causes of blindness in 552 cases in eighteen institutions for the blind in Russia, and found that in 28 per cent of the cases the blindness had resulted from smallpox. At the present day, ophthalmia neonatorum remains the most frequent cause of blindness and the most distressing, seeing that these cases must be regarded as almost entirely preventible; and the great importance of preventive treatment cannot be too strongly urged on all obstetric physicians. Among pupils in schools for the blind, who are usually under twenty years of age, we find that about 30 per cent have lost their sight from this cause (Armitage.)

Trachoma, glaucoma, sympathetic inflammation, are all very important causes of blindness, which might in many cases be prevented by timely treatment.

Home Training of the Blind.—No matter at what age blindness comes on, the sooner proper education is begun the better. This applies even to children born blind and to those losing their sight in infancy. There is a great, and perhaps not unnatural, tendency for the parents of such children to do everything for them, and to shield them from the risk of accident in every possible way, even to the extent of keeping them in bed for years. The result of such mistaken kindness is that when the child reaches school age he is defective in mind and body; he can do nothing for himself; he can hardly walk; he may have no idea of grasping, his hands and fingers hanging limp and helpless. It cannot be too strongly urged that a blind infant should, as far as possible, be treated exactly like his seeing brothers and sisters. He should be given toys to play with. He should be taught to creep and walk as soon as possible. He should be encouraged to examine everything in the room. He should be early taught to feed himself; to dress and undress. Obstacles should not be taken out of his way. He must learn by experience to avoid the furniture, to open the door, and to find his way about the house. A child brought up in this way acquires a wonderful power of avoiding objects in his path, and may come to enjoy active outdoor exercise, such as running, swimming, and skating.

There are certain bad habits which blind children are apt to develop. Chief among these are the making of grimaces and the performing of extraordinary movements. Strong sunlight apparently has a marked effect in stimulating these movements. For example, the blind person may sit upright with his fore-arms flexed, and flap his hands up and down with apparent enjoyment for hours at a stretch. Some blind people, at the same time, shake their heads from side to side. A blind child who has faint perception of light is apt to develop the habit of turning his eyes towards the sun and then spreading out the fingers of one hand and moving them up and down between his eyes and the light. Another very common habit among the blind is, when pleased with anything, to lean forward, grin horribly, and rub the hands vigorously together between the knees. Parents should be warned to look out for and check such habits at the outset. It is true that they appear to give some sort of pleasure to the child, but they are very difficult to eradicate if once formed, and are highly objectionable when the child is old enough to mix with the rest of the world.

When blindness occurs in later childhood, it is best in most cases to send the child as soon as may be to a suitable school for the blind, not only for the sake of his education, but that his interest may be aroused by the companionship of other blind pupils, and that in emulation of their acquirements he may find some solace for the loss he has sustained.

An adult suffering from any disease of the eye, likely to result ultimately in blindness, should be encouraged to begin his education at once, so that he may be in the best position possible when his sight finally fails him.

The Education of the Blind.—The idea of teaching the blind to read by means of raised characters appears to have originated in the mind of Valentin Häuy, who founded the *Institute Nationale des Jeunes Aveugles* at Paris in 1784. His example excited much interest, and before the end of the century, the Liverpool Blind School, the Edinburgh Blind Asylum, and some other schools were founded. During the present century, schools have been established in nearly all important towns, and numerous societies have been started for teaching the blind at home. Häuy made use in his books of the script form of the Roman letter. From the year

1825 to 1835 quite a number of types, all based on the Roman letter, were introduced. Of these Gall's, Howe's, Alston's, and Fry's may be mentioned. All these suffered from the disadvantage that many of the letters were not easily recognised, and consequently many blind people were unable to learn to read them.

Several stenographic and phonetic systems, such as Lucas's, were also introduced, but were discarded for school use because of their inability to teach correct spelling.

To overcome these difficulties, Moon invented a system which rapidly attained great popularity and proved most useful and practical. In his type, some of the characters suggest the ordinary Roman letters, others are arbitrary signs of his own invention. He adopted the return line of Frere, one line being read from left to right, and the next from right to left. A large number of books have been printed in Moon's type, which is still much used. The great objection to it is the bulk and costliness of the volumes. Thus the Gospel of St. John in large Moon type occupies 140 pages.

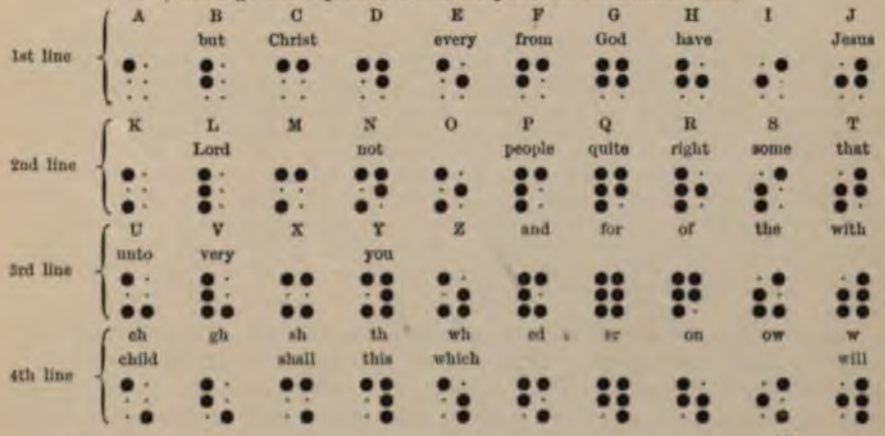
In the year 1868, the British and Foreign Blind Association was founded by a few blind gentlemen, to inquire into everything that was done for the blind, to ascertain the best methods of education and employment, and to obtain their general adoption by all means in their power. The result of their labours was the introduction into this country, and, after much opposition, the general adoption of the Braille system, invented by a blind Frenchman many years before.

The **Braille system** has many advantages. It is very simple. Writing is easily learned, so that a blind man can take notes for himself, write letters, and keep accounts. Books can be printed in comparatively small bulk, and at a comparatively low cost. Both sides of the paper can be printed on simultaneously. The system is also well adapted for musical notation.

The system is a point method, the greatest number of points of which any letter can be composed being six. These six points are arranged in upper, middle, and lower pairs. All the letters of the alphabet, punctuation marks, and several contractions can be represented by omitting one or more of these points from the group. Thus, the first ten letters, from "a" to "j," exhaust all possible combinations of the upper and middle points. The next ten, from "k" to "t," are formed from the first by adding a lower back point to each. Thus "a" becomes "k," "b" becomes "l," and so on. The third row is similarly formed by adding the second lower point.

THE BRAILLE ALPHABET AND SOME CONTRACTIONS

(The large dots represent the raised points of the Braille letter)



WRITING is carried out by means of the Braille writing-frame. This consists of a strong board with a clasp at the top for fixing the paper, and of two strips of brass hinged together which form the guides. One of these strips, which is placed underneath the paper, is marked on the surface by two rows of groups of little pits, six pits in each group, corresponding to the possible points in the letters. The second strip, which is placed over the paper, is pierced by two rows of oblong apertures corresponding to the groups of pits. Writing is carried out by means of a blunt awl, which carries a little cap of paper before it into the little pits corresponding to the letter to be formed. When the paper is turned over, these are felt as little prominences. The READING is performed from left to right, consequently the writing, being done from the back of the paper, is from right to left; but this presents no practical difficulty once the pupil has caught the idea that in reading and writing alike he has to go forward. When two lines have been written the brass guides are slipped down the paper and two more are written in the same manner.

For communicating with the outer world, Mr. Illingworth, headmaster of the Edinburgh Blind School, has recently recommended a very inexpensive typewriter which, he finds, answers the purpose satisfactorily. This is called the "Simplex," costs only 12s. 6d., and can be fitted with Braille characters, while it prints in Roman letters in ink.

For printing, thin sheets of brass are embossed either by hand, as in writing on paper, or by an ingenious machine like a large typewriter, which embosses two sheets of brass at a time. This machine has only six keys corresponding to the six points of the letters, and by pressing the appropriate keys the desired letter is formed. Pressure on a foot-lever now embosses the letter on the brass sheets. The paper to be printed is damped and placed between the two sheets of brass. These are protected by india-rubber and pressed together in a litho-press, and a beautifully clear impression is thus obtained.

ARITHMETIC is taught by means of a very ingenious frame, in which are rows of octagonal holes. A peg can be stuck into each hole in eight possible positions, thus representing the first eight numerals; 9 and 0 are represented by sticking the peg upside down in positions 1 and 2.

Various forms of kindergarten work are made use of, and bead-work is specially recommended, as the handling of small beads trains the finger-tips for afterwards feeling the dots of the Braille letters. Raised maps, models, natural history, and other specimens are all of value.

When a blind child cannot attend a special school for the blind, it is probably best for it to attend an ordinary school, where it will profit by the oral instruction, discipline, and intercourse with the seeing. If possible, some one at home should learn the Braille system, which is easily done, in order that the child may be taught to read and write. Many School Boards make special arrangements for teaching blind pupils.

Books for the Blind.—It may be of interest in conclusion to mention, as examples, the prices of one or two books for the blind, of which a large number have now been printed. All the books of the Bible are published by the British and Foreign Bible Society. These vary in price from two shillings for the Gospel according to St. Mark to five shillings for the Psalms. Nelson's *Royal Geographical Readers* are published in five volumes at about four shillings per volume. A monthly magazine, *Hora Jucunda*, is published in Edinburgh at one shilling per number. It contains sixty large pages of amusing and interesting matter, and has proved very popular. The first Braille musical monthly was started in Edinburgh in 1895 and also proved a great success. It is now published quarterly, contains twenty pages of selections from the best composers not already published in Braille, and is sold for one shilling per number.

Occupations for the Blind.—The important subject of occupations can only be dealt with very briefly. A very large number of trades have been successfully carried on by blind people, of which the chief are the making of baskets, brushes, bedding, cushions, mats, ship-fenders, ropes, sacks, chair-caning, and wood-chopping; and for girls, hand and machine sewing, netting, etc. In a workshop where the blind do not have to dispose of the goods themselves 15s. to £1 a week can usually be earned at these trades, but most institutions find it difficult to sell sufficient to give employment to all the blind seeking it. Of all occupations open to the blind, music has hitherto offered the best prospects, and as tuners, teachers, organists, etc., many blind people make a good living. In 1884, fifty-nine old pupils of the Royal Normal College and Academy of Music for the Blind, London, made a return of their earnings, which averaged over £100 per annum. Type-writing seems likely to furnish remunerative employment to a number of blind people. The phonograph is being successfully employed in lieu of shorthand notes.

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Blood.—General index of the main articles dealing with the blood, and the conditions in which it is specially affected—

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,, Pernicious.
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General Characters.—The blood may, in the first place, be regarded as a nutritive fluid for all organs which it supplies. The more highly developed the organism is, so much the more does each individual tissue and organ depend upon a sufficient and suitable supply of this fluid. One of the most characteristic properties which it possesses is its remarkable constancy of composition, considering the large amount of organic and inorganic substances that is being added to it from the side of the alimentary canal. This is brought about by a series of agencies, the intimate working of which we can in most cases only dimly surmise. They may be grouped together under the heading "selective affinity of tissues for certain substances circulating in the blood." In order that the muscles may do their work properly, they require a sufficiency of potential energy in the form of oxidisable carbohydrate, glucose. If this sugar be not withdrawn and broken down by the contractile tissues, then it becomes heaped up in the blood until its percentage rises so high that the kidneys can no longer refrain from excreting it in abnormal quantity. Under ordinary conditions, then, the muscles prevent any marked increase in the amount of glucose in the blood, presuming that the liver is not sending out so much that there is an excess left over after the tissues have used up all that they require. Not only do the muscles and contractile tissues use up the sugar, but they also, like the liver, store it up in the form of glycogen, thus preventing in yet another way the overloading of the blood with glucose. But when we consider the large quantities of sugar that are absorbed, it becomes manifest that these means of disposal are not sufficient to keep the percentage normal, and so a certain amount of glucose is transformed into fat.

Other constituents of the blood plasma, which may be formed in large amount, may have their percentage kept low by their rapid withdrawal through the kidneys, as, for example, urea. One of the best examples of an organ which exercises the property of selective affinity to a very marked degree is the mammary gland. The researches of Bunge and his pupils have recently taught us that every animal secretes a milk most suitable for the needs of its young offspring. If the young animal require much lime it obtains it, although the percentage in the blood be very small. The mammary gland selects from the plasma what is requisite for the nutrition of the young organism. From the substances so picked out, by a marvellous process of elaboration and synthesis, the specific milk constituents are formed. This is not the place to speak of the rôle of proteids in metabolism, but with regard to the point at present under discussion, it is important to remember that although the amount of albuminous substances digested is so great, there is a remarkable constancy in their amount in the blood.

For the building up of the young and the maintenance of the adult organism, these organic substances are always in request. When their amount in the food is diminished to such an extent that starvation arises, then the higher tissues live at the expense of the proteids of the lower ones. At any special time in the developmental life, when a particular organ requires special sustenance, it may derive it from the organic constituents of some other tissue. Miescher described a most remarkable instance of this in the growth of the ovaries in the salmon at the expense of particular muscles during the time when no food was being taken in.

The form in which the proteid leaves the one organ to be transmitted by means of the blood to the other one may not be that in which it is stored up in the latter. The receiving organ may, and in many cases does, take up from the blood more than one constituent, and by manifold syntheses builds up the substances that it requires. For example, in the salmon the formation of vitellins in the ova goes on at the expense of the globulins derived from degenerating muscles. Every organ stretches out its hands for its particular food requirements to the blood, and gives off to the same fluid what has been rendered useless and effete or what is required for some other tissue or organ.

We have seen that the blood remains remarkably constant with regard to the constituents necessary for the nutrition of the tissues; but perhaps in no particular is this constancy more marked than in the maintenance of its normal alkalinity. In order that each cell in the body may perform its functions, the alkalinity of the blood and the fluids derived from it must be kept up. One cannot say simply that the alkalinity is necessary for oxidation, because the carbonate or bi-carbonate in the blood will not aid oxidation to any marked extent. It is more as the carrier of the carbonic acid from the tissues that the carbonate is required. The oxidation processes occur outside the blood by means of the tissues. Researches carried out some years ago showed how the organism resisted any tendency to alteration in the reaction of the blood. It was clearly demonstrated that mineral acids, on being given to omnivora or carnivora, did not diminish its alkalinity, this being avoided by the tissues breaking down to furnish ammonia sufficient to neutralise the acid and so spare the alkali of the blood. In herbivora, where the protective action from the side of the tissues does not come into play, acids act as poisons. In certain forms of poisoning, where there is an increased breaking down of proteid with a rise in the amount of sarcolactic acid, the ammonia derived from the tissues again steps in to spare the alkali of the blood.

Before we go on to the study of the rôle that the cells of the blood play in vital processes, there is another important property of the serum which requires to be referred to, namely, the protection which it affords, to the organism under certain conditions, against the action of many bacteria and their products. A particular species or even an individual may be refractory to one particular infectious disease; or the condition of immunity in a susceptible individual may be attained by the introduction of the blood serum from an animal which has been rendered artificially immune to that disease. For example, it is the cell-free blood serum of animals rendered immune to tetanus which destroys the toxic substances produced by the tetanus bacillus. So also in the natural recovery from an infectious disease, there are formed in the body substances which are bactericidal to the specific organism of that disease, or which neutralise the poisonous products of that organism. With regard to the chemical nature of the substances which give the serum this antitoxic property, we know but little, and the same is true with regard to their place and mode of formation. Recent work on pneumococcal infection points to the probability that the bone marrow is one of the seats of formation of the immunising substance. Whether it be a product of the leucocytes or not, it is at present impossible to say.

There is another point of importance in the physiology of the blood which I shall now refer to. One must bear in mind that the way in which the different inorganic and organic constituents are combined in the circulating blood must be very different from that which we find to occur in the blood when examined after death. The albumin and globulin do not circulate free in the plasma, they are in all probability more or less loosely bound to many of the salts which are present. They undoubtedly play a part in the transmission of the gases in the blood. The greater or less firmness with which sodium chloride is united to serum globulin may have to do with a decrease or increase in the amount of salt retained in the plasma, and hence a corresponding alteration in its amount in the urine.

Up to the present, reference has exclusively been made to the plasma and its constituents; I shall now very shortly refer to the importance of the solid elements, a description of the general form and structure of the red and white cells being given later. The most important character of the *red cells* is that they contain the oxygen-carrier of the blood, hæmoglobin; but we must not forget that there are other proteids present in these cells which also play a distinctive rôle in their life-history, viz. those present in the stroma. Of special interest in this respect are the alterations in the form of the red cells in and outside the body, all pointing to their great elasticity. They are also resistant bodies, partly physically in virtue of their elasticity, and partly vitally, preventing, for example, diffusion inwards of solutions of iron and manganese, although permitting glucose readily to pass in. Alterations in the form of these cells by increasing or decreasing the surface area with or without alteration in their volume may lead to a disturbance in their respiratory capacity. Under certain conditions, to be afterwards described, the staining reactions of the protoplasm may be altered, while under other conditions nucleated forms (erythroblasts) may appear in the circulating blood.

The white cells, although much less numerous than the red ones, have also important functions to perform, and in disease constitute a more important element than the latter. They are assimilative organisms, and affect by alterations in their metabolism the general body metabolism. The fact that certain of them show marked phagocytic properties has resulted in the term "blood scavengers" being applied to them. But they are more than destroyers of what is effete, they are also secretory cells, as shown especially in the granular forms, where undoubtedly the production of substances essential for the general body welfare goes on.

A. CORPUSCULAR ELEMENTS AND GRANULES

RED BLOOD CORPUSCLES or ERYTHROCYTES are small, round, non-nucleated discs, which are thinner in the centre than at the periphery, so that when seen lying edgewise they show a distinct indentation on both sides. On microscopic examination the convex periphery is first of all brought into focus, and only later are the rays passing through the centre intercepted by the objective. They are of light yellow tint, with a slight admixture of green, and when examined quickly in a fresh specimen are seen to have a smooth contour, and to be soft and elastic. When the cover-glass is tapped, they roll across the field, altering their course when they come in contact with a white corpuscle. Very shortly afterwards, they begin to run together to form smaller or larger rouleaux. Those at the edge of the preparation begin rapidly to show irregularity of outline, and, as drying proceeds, all become distinctly crenated. This shrinkage of these elastic cells is due to the increased concentration of the plasma from evaporation, water passing from the corpuscles to the denser salt solution outside. The average diameter of these cells is $7 \cdot 5\ \mu$; only rarely are smaller or larger forms to be met with in normal blood. A great deal of caution has to be observed in speaking of the minute structure of these cells, as it is extremely difficult to distinguish artificial and degenerative appearances from normal ones in stained films. The older views simply referred to the stroma with the hæmoglobin contained in it, the "oikoid" and "zoöid" of Brücke. Recent work is supporting the view that a peripheral can be distinguished from a central part, the former containing the mass of the hæmoglobin, the latter the residue of the degenerated nuclear matter of the erythroblast—"nucleoid." It is important to remember that the peripheral part stains most deeply with eosin, partly because it contains more hæmoglobin. In pathological conditions, when the red cells do not stain so deeply with eosin as normally, this diminution is not due to an altered character of the hæmoglobin, but to a decrease in its amount.

The *form* of the cells may be markedly altered in various diseases, the most important variations being found in the anæmias, in the condition known as poikilocytosis. Cells of normal size are to be seen along with large and small ones, all except the smallest showing the distinct dell in the centre. There are also to be seen a variety of the most bizarre forms, some presenting the appearance of tiny pears, others kidney-shaped or oval in shape. The smallest forms, the microcytes, are believed by Ehrlich to be due to contraction of the poikilocytes, that is to say, analogous to the crenated forms of the erythrocytes. This, however, is doubtful, though it is possible that they are degeneration forms of the erythrocytes resulting from a segmentation of the latter.

In addition to the alterations in form, there are also variations *in staining properties* to be noticed in different pathological conditions. Reference has already been made to alterations of a quantitative type in eosin-stained preparations, but the anæmic degenerations require to be discussed more fully. In a dried film the reds take up the eosin out of an eosin-hæmatoxylin mixture and stain of a pure red tint, while in certain forms of anæmia more or less of the hæmatoxylin is also taken up, so that the cells take on a bluish tint. One may speak of these corpuscles, then, as polychromatophil. This is undoubtedly due to the fact that the cells are no longer so resistant to the entrance of the hæmatoxylin which now diffuses inwards. It cannot be due to an alteration in the character of the blood pigment. Frequently in anæmic conditions a simple diminution in the amount of hæmoglobin in each cell is seen from the light tint after staining with eosin. Even in normal blood both of these qualitative and quantitative alterations may be seen to a slight degree, as one would naturally expect, seeing that the cells are being constantly exposed to alterations in the plasma. Care of course has to be taken to exclude changes due to different durations of staining and insufficient fixation.

Another pathological alteration in the red cells is the appearance of *nucleated forms* (erythroblasts) of different size—normoblasts, megaloblasts, and microblasts. The first of these three are the forerunners of the normal erythrocytes. Under ordinary conditions they do not pass into the blood, but are always to be found in the bone marrow of the adult. Megaloblasts are only very rarely found in the adult marrow, but are present in that of the fœtus, and also for the first few years after birth. *The normoblasts* contain a nucleus which stains very deeply and homogeneously with nuclear dyes, and presents a well-defined contour. Very frequently it is found lying slightly excentrically in the cell. There may be more than one nucleus present. The cytoplasm usually stains in the pure hæmoglobin tint, but there are frequently qualitative alterations of the polychromatophil type.

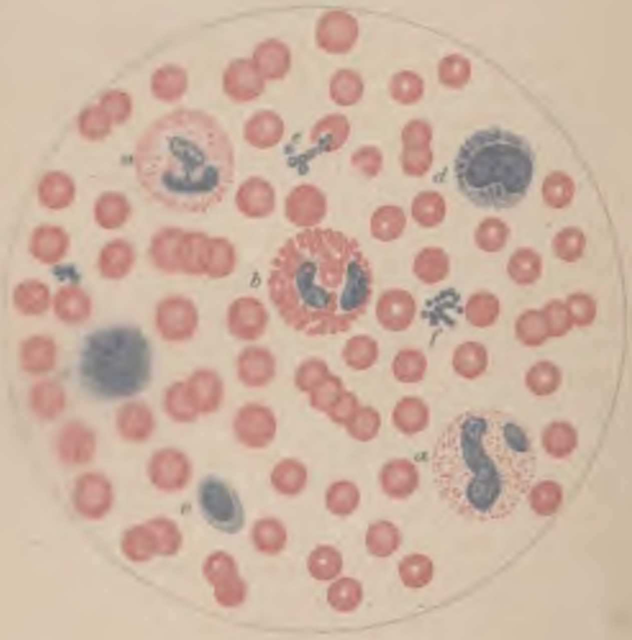
The megaloblasts are always much larger than the normoblasts, frequently being four times their size. The nucleus, although larger than that of the normoblast, is, in comparison to their respective cytoplasms, usually smaller than in the latter. The staining affinities of the nuclei in these large cells vary, always less, however, than those of the normoblasts.

The cytoplasm of those cells is more frequently polychromatophil than that of the normoblasts.

The small nucleated forms—*microblasts*—are rare, and, so far as is at present known, of no special pathological importance.

The *number* of the red cells may also undergo variations. Here only a few points of importance need be referred to, as the subject is discussed in other articles. One may say that in the vast majority of cases an increase in number is due to a decrease in the amount of plasma. Therefore when polycythæmia is referred to, it is, as a rule, a relative, not an absolute, increase of the total red corpuscles in the circulating blood. They may undergo variations in number in different physiological conditions (dietetic, climatic, rapid or slow functional alterations as at birth), pathological (primary and secondary anæmias in different cachectic conditions), and also from action of poisons or drugs (lead, iron, arsenic, mercury, and all agents which act as disturbers of the amount of plasma, such as purgatives). The term "polycythæmia" is applied to those conditions where the number of red cells per unit volume of blood is higher than the normal, while "oligocythæmia" signifies a decrease per unit volume. The variations in the number of the red cells *per se* are not so important as alterations in the amount of hæmoglobin in each cell. Oligocythæmia may be artificially produced in animals by withdrawal of varying quantities of blood. Almost immediately after the removal, the blood-pressure begins to sink, but in a very short time it again commences slowly to rise, until it becomes almost as high as at first. The reason for this rise is twofold. In the first place, there is the vaso-motor action trying to counteract the effect of the greater emptiness of the vessels; and secondly, there is the passage inwards of lymph into the blood to supply the place of the fluid that has been lost. This latter action takes place when the vaso-motor effect is passing off. Pathologically, oligocythæmia may be produced in a variety of ways. There is frequently a diminution in the formation of red cells in the primary anæmias, or there may be an increased breaking down of the corpuscles, as in pernicious anæmia, or an increased passage inwards of lymph, as in recovery after hæmorrhage. The great diversity of opinion existing as to the cause of oligo- or polycythæmia is probably due to the fact that in a large number of conditions (physiological as well as pathological) the variations are not due to the disturbance of one etiological factor, but to many. To take an example, the undoubted increase in red cells that takes place when an animal is taken from a low to a high altitude is probably due partly to an increase in the removal of water from the body, partly to their altered vascular distribution, and in part to increased activity of bone marrow, as shown by the appearance of young red cells in the blood. Miescher believed that this latter cause was an important one. He emphasised the lowered oxygen tension in the blood as the specific formative stimulus. At the commencement of residence at high altitudes there is no increase either in the amount of hæmoglobin or in the total volume of the red cells, there is simply a greater surface area of hæmoglobin-holding cells exposed in order to accommodate the organism to the low atmospheric pressure. One must remember that under these conditions other etiological factors come into play; for example, the action of the intense light, increased rapidity of respiration, and increase in muscular exercise. It seems most likely that after prolonged residence at high altitudes there is an absolute increase in the total number of the red cells, at the outset simply a relative polycythæmia due to increased water excretion.

White Blood Corpuscles.—These cells are from many points of view the most important ones in the blood, as variations in their number or characters are frequently the most distinctive signs of pathological variations in the blood-formative parts. At no time has a greater diversity of opinion existed than at present as to the proper classification of those occurring normally in the blood or passing into it owing to changes in the blood itself or in the marrow or organs containing lymphoid tissue. A very prevalent view at the present day is that *all forms of the white corpuscles arise from the small lymphocytes*, and that transition forms, showing the necessary alterations in nuclear structure and cytoplasm, can be distinguished between these cells and the so-called fully-formed polymorphic neutrophilous leucocyte. Others hold that the *lymphocytes and the granular cells have in adult life a separate origin and life-history*, that the adult lymphocyte has no connection with the polymorphic granular leucocyte. Here a very short description will be given of the different types



NORMAL BLOOD.

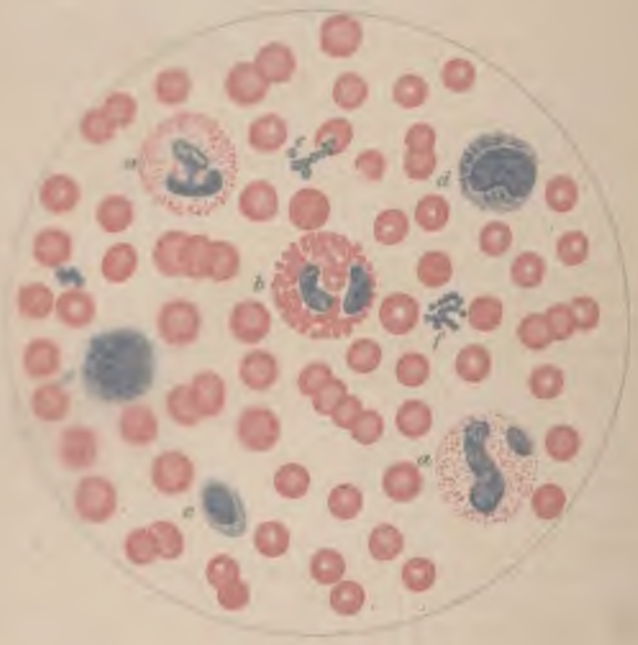
Film preparation fixed by heat; stained with 1 per cent. watery eosin for ten minutes; washed and stained with Löffler's methylene-blue for twenty to thirty seconds; washed, dried, and mounted in Canada balsam

Magnified 1000 diameters.

a = Polymorphic neutrophilous leucocytes.
b = Coarse eosinophilous polymorphic ~~neutrophilous~~ leucocytes.
c = Small lymphocyte.
d = Large lymphocytes.
e = Blood plates leaving an erythrocyte, or adherent to it.
f = Accumulation of blood plates.

The small round deeply stained cells remaining are the erythrocytes.

NOTE.—*Although these cells were drawn from an actual preparation, they did not all occur in one field as shown here.*



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to be met with in the normal blood of the adult, then those which pass in under different pathological conditions; and finally, when speaking of the development of these cells, a brief survey will be taken of the views in support of the two theories just referred to.

1. *Small lymphocytes.*—These are small cells, as a rule about the size of a normal red corpuscle, containing a large, round, usually centrally placed nucleus which stains more or less homogeneously. The zone of protoplasm around the nucleus is very small in amount, and frequently difficult to make out. The cytoplasm after the action of some stains seems more basophil than the nucleus. These cells are not amœboid.

2. *Large lymphocytes* or hyaline cells have many points in common with the former, and represent in all probability their more adult forms. They are usually twice or three times the size of the erythrocytes, and contain a large nucleus which is poorer in chromation than that of the small lymphocyte, and which frequently shows indentation or even fragmentation. The protoplasm reacts strongly alkaline to erythrosin. There is never so distinct granulation in these cells as occurs in the granular forms, although the cytoplasm frequently shows differentiation in preparations stained with methylene blue, probably due to the spongioplastic network. Frequently the zone of protoplasm around the nucleus is irregular, and may show signs of degeneration. As far as can be made out, the life-history of this cell ends with the fragmentation of its nucleus. It is exceedingly difficult to find any sign of a definite transition between this cell and the polymorphic neutrophilous. The properties of the chromatin and the cytoplasm in the two forms are so different that it is difficult to imagine a transition of the one into the other.

3. There is a type of cell, especially referred to by Ehrlich, which is difficult to characterise both as to its relationship to the other forms and to its place of origin, namely, THE LARGE MONONUCLEATED LEUCOCYTE. It is about the same size as the large lymphocyte, but possesses more protoplasm which contains no granules, but is always distinctly less basophil than that of the lymphocyte. The nucleus is round or oval, and stains faintly with nuclear dyes. It undergoes indentation, then the chromatin becomes denser, and finally neutrophilous granules begin to appear in the cytoplasm, which gradually loses its basophil properties. This cell is almost certainly formed in the bone marrow.

4. *Polymorphic* (*Polymorphonuclear*) *Neutrophilous Leucocytes.*— These constitute the largest proportion of the leucocytes present in normal blood. They are undoubtedly for the most part myelogenic. There are two distinct points which characterise these cells: in the first place, the peculiarly irregular arrangement of the chromatin, the nucleus presenting a nodulated appearance, frequently in the form of an irregular *E*, *S*, or *Z*; and secondly, the appearance of very numerous neutrophilous or finely eosinophilous granules. The nucleus may break down into two or more fragments, rendering the cell polynuclear. In only the rarest instances (and even these not sufficiently well defined) have other types of granules, such as coarse eosinophilous, been described as occurring along with the neutrophilous. The nucleus is practically always rich in chromatin. These are the typical amœboid leucocytes which wander out into the tissues in acute inflammation and go to form the pus corpuscles.

5. *The true eosinophilous leucocytes* are characterised by their coarse, highly refractile granules, which are, in man at least, practically always round and of regular contour. These stain more or less homogeneously with acid anilin dyes, such as eosin. The nucleus of this cell is similar to that

of the neutrophilous, but as a rule takes on the basic stain less deeply. The cell is amœboid, only wandering out in all probability when attracted by a specific stimulus.

6. *Basophilous Leucocytes—Mast cells*—are only found in exceedingly small numbers in the normal blood. The nuclei are very poor in chromatin, and their granules, which vary in size, stain of a violet tint with methylene blue. They are increased in the blood in leucocythæmia.

The following table gives what may be regarded as approximately the average percentage of these different forms of white blood corpuscles found in the normal blood.

1. Lymphocytes (small and large)	20-25 per cent.
2. Large mononucleated leucocytes and transition forms	2·5 "
3. Polymorphic (polynuclear) neutrophilous or finely eosinophilous	70 "
4. True eosinophilous polymorphic (coarsely granular)	3 "
5. Mast cells . . . barely	0·5 "

It is, however, by no means rare to have alterations in the relative numbers of these cells under normal conditions in different individuals. I have noticed that this very often occurs in the case of the eosinophilous.

There are certain other forms which *only occur pathologically in the blood*, and require to be merely briefly referred to here, as their nature and significance are discussed in other articles.

1. *Neutrophilous Myelocytes.*—These are mononucleated cells with a relatively large, faintly stainable nucleus and a granular protoplasm fairly regularly arranged around it. The granules are neutrophilous or faintly eosinophilous. These cells are on an average distinctly larger than the polymorphic neutrophilous, often about 16 μ in diameter, but forms even slightly smaller than the erythrocyte are also to be met with. Most authorities hold that these cells are not amœboid. They are of great pathological importance, occurring especially in the blood in medullary leucocythæmia, but they are also present in other conditions, for example, in certain diseases of children, in diphtheria, and about the time of the crisis in pneumonia.

2. *Eosinophilous Myelocytes.*—These are large mononucleated cells similar to the neutrophilous, except that the granules present in them are coarser and have a more marked affinity for eosin.

There are other cells which have been described as occurring in the blood in different pathological conditions, the nature of which, however, is still so doubtful that it is better not to describe them in this short article. Very many varieties of myelocytes in different transition changes are to be seen in the blood in medullary leucocythæmia.

BLOOD PLATES.—These exceedingly interesting structures seem undoubtedly to be preformed in the circulating blood, although this is not believed by some investigators (Löwit). Their form is easily altered by the action of different agents; normally, however, they are more or less rounded and about 3 μ in diameter. As a rule they do not contain hæmoglobin, although some undoubtedly do. Chemically they are found to be rich in phosphorus, so that, bearing in mind their staining affinities, there is strong proof that they are nuclear or nucleoid in type. Bizzozero is of the opinion that they are independent bodies and have no relationship to either the white or red cells. A very widespread view is that they are derived from a fragmentation of the nucleus of the leucocyte, although there is no very good proof of this.

Hayem and his pupils believe that they are the forerunners of the red corpuscles and so term them hæmatoblasts, a view that is practically entirely abandoned at the present day. Recent work is tending to show that they may be derived from the residue of nuclear matter left in the adult erythrocyte. There seems to be an intimate connection between the number of blood plates and the rapidity of blood-clotting, because in conditions where they are decidedly increased in number, *e.g.* chlorosis and in the anæmia after prolonged hæmorrhage, the rate at which coagulation occurs is also increased, while in purpura, where clotting is very slow, their number is small. Arnold has recently described the separation of these bodies from the erythrocytes in the vessels of the mesentery of young mice during life. As far as could be made out, the leucocytes were not implicated in the process. It is difficult to study in blood films the relationship between red cells and blood plates, owing to the tendency of the latter to adhere to the former.

Granular Bodies.—Small round, highly refractile granules are frequently to be seen in normal and pathological blood. They undoubtedly include substances of different type and origin,—granular bodies derived from leucocytes, from degenerating red cells and blood plates, and also from precipitates in the blood plasma. They have been termed by Müller, "Hæmokonien" or "Blutstäubchen" (blood dust particles).

Chemical Composition — 1. Red Cells. — The main constituents are hæmoglobin, a globulin which coagulates at 75° C., lecithin, cholesterin, and salts of potassium, sodium, iron, calcium and magnesium. The most important salts are the chlorides and phosphates. There is also about 70 per cent water. By far the most important constituent is the hæmoglobin, and it is with variations in the amount of this body that the clinician has mainly to deal. Most of the constituents are in a labile state, and this especially holds good of the soluble inorganic salts, *e.g.* the chlorides. The percentages of water and chlorides go hand in hand. The organic nitrogenous constituents are not nearly so unstable, the interchange between plasma and cells depending largely upon their state of solution with the salts present. The stroma of the cells is albuminous in nature. A point of great importance, and one that is apt to be overlooked, is that in the non-nucleated mammalian erythrocyte there is probably still some nuclear matter present which, under the influence of different agents, leaves the cell in the form of the mother substance of the blood plates (*see* Plate).

Analyses have been made of the human red blood corpuscles, but the results are unsatisfactory for the reason that after their separation it is impossible to wash them free from the constituents of the plasma without removing in part, at least, some of the more soluble constituents of the cells.

2. Leucocytes.—As it is impossible to separate those cells so as to admit of an exact chemical analysis, most of our knowledge as to their composition is based upon results derived from examination of pus, organs rich in lymphocytes and leucocythæmic blood. A few points of great interest in their chemical composition have been made out by Kossel and others, supplementing what we already know from the microscopic examination. They are rich in a complex proteid, nucleo-histon, which is composed of nuclein combined with a body possessing many of the characters of an albumose, namely, histon. The high phosphorus percentage of the cells depends mainly upon the presence of this substance and lecithin. Some of the other important constituents are derived from the nucleo-histon,

namely, the alloxur bases—adenin, guanin, xanthin. Glycogen is present in some, if not all, of the cells, as are also cholesterin, lecithin, and fats. The inorganic salts are the same as those met with in the red blood corpuscles. The specific granules in the cells are probably either nucleo-histon or derivatives from that body.

3. Blood Plates.—Reference has already been made to their chemical composition. They undoubtedly contain organically formed phosphorus, and are only with great difficulty dissolved by the action of the gastric juice. They contain glycogen. Their probable nucleoid nature has been sufficiently referred to.

4. Granules.—Some are fatty, others albuminous, and many are pigment granules derived from breaking down of hæmoglobin.

Developmental History.—It would not be advisable to give here more than the briefest account of the development of the blood corpuscles. The most diverse opinions are held at the present day even on such fundamental points as the seats of formation of these cells in fœtal and adult life. One of the most important contributions to our knowledge of this subject which has been made within recent years is that of Saxer. He noticed that wandering cells, each possessing a large round nucleus and finely granular protoplasm, are to be found in the connective tissue very early in embryonic life (sheep embryos 9 mm. long). These differed from the fixed connective tissue cells in possessing a large nucleus and no clasping processes. In larger embryos (25 mm. long) groups of these cells are seen lying close to the blood-vessels, but independent of them. They are seen to divide mitotically and produce smaller cells, which possess small round nuclei, similar to those of the erythroblasts, and also contain hæmoglobin deposited in the cytoplasm. Giant cells are also to be seen containing in their protoplasm degenerating red cells. In still older embryos leucocytes appear for the first time, lying close to the lymphatics. They arise from the same cells as the red ones which have just been described. Later on in embryonic life (embryos 13·5 cm. long) the formation of red cells in the connective tissue stops, the liver, spleen, lymphatic glands, and bone marrow, as their development proceeds, taking on the function instead. The liver is the most important seat of formation of the erythroblasts in embryonic life. It is very doubtful, however, whether there is at any time an endogenous formation of nucleated or non-nucleated red blood corpuscles in giant cells or vessel-forming cells (Ranvier) in the liver or elsewhere. Normally in man, at least, there is no formation of erythroblasts after birth in liver, spleen, or lymphatic glands, the bone marrow then being the sole seat of their development. It is difficult to say whether there is a hæmoglobin-free forerunner of the erythroblasts occurring at the centres of development of the latter. The mother cell—Saxer's wandering cell—is, of course, free from hæmoglobin, but in the later development of erythroblasts it is difficult to be certain whether there is a cell free from hæmoglobin, having the general characters of the erythroblast, which is transformed into the typical pigmented cell. There seems undoubtedly to be an extra- as well as intra-vascular development of erythroblasts in the bone marrow. The forerunners of the granular leucocytes are developed outside the vessels of the marrow. These developmental changes are seen in the red marrow both in embryonic life and after birth. In pathological conditions erythroblasts may be found in the spleen in adult life, as, for example, in leucocythæmia. The mode of transformation of the nucleated red blood corpuscle (erythroblast) into the non-nucleated one (erythrocyte) is still a subject of controversy. Some believe that the nucleus leaves the cell by a process of extrusion, basing their opinion upon the fact that transition changes can be seen showing the gradual passage outwards of the nucleus. Others hold that the nuclear matter breaks down and dissolves in the cell (karyolysis), and they describe appearances, especially in preparations stained with methylene blue, that seem to support their view. They affirm that extrusion of the nuclei, when it occurs, is artificial. Ehrlich believes that the first view is true for the normoblasts, and the second for the megaloblasts. It seems likely that the nuclear matter does undergo at least partial degeneration, before it is extruded, and thus some nucleoid matter may be left in the cell.

With regard to the *white blood corpuscles*, it is most important to know their centres of formation after birth. In adult life the seats of formation are the same as in embryonic life, except that the thymus gland after birth gradually becomes inactive. There are three great developmental centres, lymphatic glands (including all collections of lymphoid tissue), spleen, and bone marrow. Many hold that the lymphocytes are the forerunners of all the other forms, and in early embryonic life this may be true; but the view held by another section has much to support it, namely, that the granular cells (myelocytes) formed in the bone marrow are the forerunners of the true granular cells of the blood, while the lymphocytes which are formed in the germinal centres of the spleen and lymphatic glands pass into the circulating blood in the same form, and do not develop into the granular leucocytes. The nuclei of the large lymphocytes may undergo indentation and finally fragmentation without any appearance of neutrophilous granules in the cytoplasm. There is no doubt that the lymphocyte may at least pass through its developmental course without passing through a stage similar to the neutrophilous polymorphic leucocytes.

Much can be said in support of each view, but one may say that our fuller knowledge of the two great types of leucocythæmia has strengthened the latter view. One cannot draw definite conclusions simply from seeing cells that might possibly be transition forms between lymphocytes and granular leucocytes. Judgment must be based upon results of experiment, such as removal of spleen, pathological lesions of the blood-formative parts (*e.g.* of bone marrow, spleen, lymphoid tissues) and developmental history. The subject is still, however, *sub judice*.

PHYSIOLOGICAL AND PATHOLOGICAL VARIATIONS.—Those affecting the red cells have already been mentioned, while the main alterations in the leucocytes will be referred to in other articles (see leucocytosis, leucocythæmia). Here a reference will simply be made to some of the conditions leading to an increase or a decrease in their numbers. An increase affecting practically all the varieties of white cells occurs in different physiological conditions, for example, in the *digestion leucocytosis*, occurring about four hours after a meal, in the *newly born* and in *pregnancy*. Two great types of leucocytosis may be distinguished, namely, one affecting especially or entirely the non-granular cells (passive leucocytosis, *i.e.* an increase in the non-amœboid cells, the lymphocytes), and the other affecting the granular forms, either those which are present normally in the blood, or those which, under normal conditions, do not leave the marrow, the myelocytes (active leucocytosis, *i.e.* of the amœboid cells or their forerunners).

I. *Lymphocytosis* (*Passive Leucocytosis*).—By far the most important condition in which this occurs is the lymphatic form of leucocythæmia; but a relative lymphocytosis occurs also in children's diseases, such as rickets and whooping-cough, and also in hereditary syphilis. It occasionally occurs in secondary anæmia in the adult.

II. *Active Leucocytosis.*—The most common form is the polymorphic neutrophilous leucocytosis, where the eosinophilous cells and lymphocytes may be both relatively and absolutely diminished. This occurs under different physiological and pathological conditions, especially well marked in the infectious diseases.

By an eosinophilous leucocytosis one means both a relative and absolute increase in the true eosinophilous cells. The most important pathological conditions in which this occurs are asthma, certain skin affections, and in helminthiasis. A very important point in these conditions is the appearance of Charcot-Leyden crystals in fæces or sputum. The views that are held as to the mode of production of these different forms will be discussed in the article on leucocytosis.

The passage of myelocytes from the marrow into the blood occurs most markedly in medullary leucocythæmia.

III. *Leucopenia* or diminution in the number of the white corpuscles is usually due simply to an altered distribution of the cells, as in the preliminary hypoleucocytosis after injection of animal extracts, when the leucocytes have been withdrawn to the pulmonary capillaries, and are there heaped up. A diminution due to an increased breaking down of the cells must be rare. The decrease in the numbers in the blood may be due to a diminution in their withdrawal from the marrow or from a decreased functional activity of the same. In some cases, after injection of nucleic acid there seems to be, in comparison with the normal, an increased breaking down of leucocytes (leucolysis), or an increase in the metabolic activity of those cells accompanying the leucocytosis. This has been inferred from the increased P_2O_5 excretion in the leucocytosis after nucleic acid.[1]

The blood plates are markedly diminished in pernicious anæmia. Other conditions in which their numbers vary have been already referred to.

B. Plasma and Serum

These may be taken together, as they contain to a very large extent the same constituents, the principal difference being that the fibrinogen has been removed from the plasma in the formation of the serum. The three principal albuminous substances in the plasma are the serum albumin, serum globulin, and fibrinogen. Under certain pathological conditions albumose and perhaps also true peptone may be present. The causes leading to the appearance of these two bodies are obscure. In some cases there is probably an alteration in the intestinal mucous membrane, allowing them to pass through without being synthetised to albumin; in others they are probably derived from pus breaking down somewhere in the body, while last of all they may be obtained from intracellular digestion. The most important pathological condition in which they occur in the blood is leucocythæmia. As emphasised in the introduction to this article, the total amount of proteids in the plasma is very constant; but the serum globulin may be increased without the albumin, or at the expense even of the latter. For example, in some forms of nephritis the ratio of albumin to globulin, instead of being 1 : 1·5, is 1 : 11·5. In these cases a large amount of albumin diffuses out through the kidneys. With increase in blood-pressure albumin passes out more readily than globulin. The most important salts are carbonates, phosphates, and chlorides. The phosphates in the plasma are increased in conditions where there is a rise in the metabolism of the nucleated elements of the blood, as in different forms of experimental leucocytosis.

Nature and Rapidity of Coagulation.—*Extra-vascular Coagulation.*—When blood leaves the vessels, it is transformed within a few minutes into a more or less gelatinous coagulum. *Intra-vascular clotting* also occurs when the vessel walls undergo certain pathological alterations. It is contact with the uninjured vessel wall that prevents coagulation, as was long ago pointed out by Hewson and Brücke. Pathologically, however, it may occur within vessels that have undergone no change, as, for example, after the action of certain agents, such as pyrogallol and the chlorates of the alkalies.

There can be no doubt that *changes in the leucocytes* play some part in the causation of blood-clotting. A fluid which contains fibrinogen cannot undergo coagulation unless one containing leucocytes or fibrin ferment be added. Perhaps the most widespread view at the present day is that, before the transformation of fibrinogen into fibrin can occur, the leucocytes commence to break down and set free the zymogen or forerunner of the fibrin ferment. This body may be derived from either the nucleus or the cytoplasm, perhaps from the granules in the latter. It seems unnecessary that the cells should undergo an actual degeneration. In solutions of fibrinogen which contain no soluble lime salts, this zymogen is unable to act. It is important to bear in mind that the calcium salt does not act as a transformer of soluble fibrinogen into insoluble fibrin, but of the zymogen into the true fibrin ferment. Fibrin contains no more calcium, weight for weight, than fibrinogen.

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[1] Milroy and Malcolm, *Journ. of Physiology*, July 1898.

It is difficult at present to define the *part which the red cells play* in the process, but there are strong grounds in support of their active participation. Reference has already been made to the relationship that probably exists between the blood plates and degenerative changes in the reds, and also to the connection between the number of the former and the rate of coagulation. As Arnold has pointed out, the formation of *blood plates* is especially well seen in the early stages of intra-vascular clotting.

The *rate of coagulation* varies in different physiological and pathological conditions, *e.g.* it may be delayed to a slight or marked degree in inflammatory conditions, and also especially in scurvy and hæmophilia. Usually distinct signs of fibrin formation occur one and a half minutes after the blood has been shed, but a long time elapses (12-48 hours) before complete separation of the clot and serum takes place. In some cases this separation may be very imperfect even when coagulation has not been markedly delayed, as Hayem has especially emphasised in the case of pernicious anæmia. In certain animals, such as the horse, and in pathological cases where there is delay in the process of coagulation, the clot shows an upper gray or buffy coat and a lower less compact red one, the latter composed of the heavier red cells with an interlacing fibrin network, the upper mainly of white blood corpuscles with fibrin. The process may be prevented by not allowing the fibrin ferment to be formed, as, for example, after withdrawal of soluble lime salts by ammonium oxalate. Injection of albumose, peptone, leech extract (obtained from anterior parts of animal), and salts of the bile acids all delay coagulation, and may prevent it altogether.

METHODS OF CLINICAL EXAMINATION

A. GENERAL

I. ESTIMATION OF TOTAL QUANTITY.—Many methods have been devised for this purpose, but all of them are more or less fallacious. The more important methods only will be described.

Quincke's Method.—The red corpuscles in a certain volume of blood are numbered. Then shortly after transfusion of a known quantity of blood in which the number of red cells has been estimated, the corpuscles are again numbered. After uniform distribution of the red corpuscles, their increase per unit volume would allow one to calculate the total amount of blood present. The method is, in the first place, troublesome to carry out, and it requires the injection of large quantities of blood in order to produce any appreciable effect. Of course there are also the two great fallacies, alterations in the transudation of plasma and breaking down of the red corpuscles.

A Similar Method was introduced by Vierordt.—A measured quantity of blood is withdrawn and the red cells in it estimated, then (time being allowed to elapse for the return of the blood to its original volume by absorption of lymph without any new formation of red cells) another portion is withdrawn and the number of red cells in it enumerated. The total volume of blood is then easily estimated, thus—

If α = number of red cells in the first portion of blood withdrawn;
If β = number in second portion;
And γ = volume of blood withdrawn in first portion;

Then total quantity $= \frac{\alpha\gamma}{\beta - \gamma}$.

The method recommended by *Ehrlich* has much in its favour. He advises the estimation of the degree of dilution that a known quantity of antitoxin of fixed strength undergoes after injection into the blood-stream, the blood being examined again, after some time has elapsed, as to its antitoxic power.

No method is suitable, however, for clinical work, all being very troublesome and more or less fallacious.

II. ESTIMATION OF THE VOLUME OF CORPUSCLES AND PLASMA.—The method which has been largely used for this purpose is so fallacious that the pains spent in carrying it out are practically thrown away. The method referred to is the estimation of the volume of the corpuscles after centrifugalising a known volume of diluted blood. The apparatus most commonly used is one or other modification of Hedin's hæmatocrit.

It is practically impossible to get a suitable diluting fluid that does not injure the corpuscles, so that, bearing in mind the extremely small quantity of undiluted blood used and the fact that the corpuscles are compressible, it can easily be understood that the method leaves much to be desired in the matter of accuracy. Full descriptions of the instrument and the method of its employment are given in von Limbeck's and Cabot's works.

III. ESTIMATION OF SPECIFIC GRAVITY.—Here also the methods are so troublesome, and the results obtained of so doubtful clinical importance, that they are at present but seldom carried out. The most exact methods are those based upon determining the weight of a given volume of blood by means of the capillary pyknometer (Schmaltz's method).

A thin-walled capillary glass tube, about 12 c.m. long, after being washed out with water, alcohol, and ether, is dried and weighed. It is then filled with distilled water and again weighed. The water is then blown out, the tube washed with alcohol and ether, and the blood sucked up until it is full, when the weight is again taken. Corrections for temperature may be made, though they are unnecessary. By subtracting the weight of the tube from that of tube plus water in the one case, and from tube plus blood in the other, one obtains the specific weight of the latter when water is taken as unity.

The tendency for the blood to coagulate in the small tube occasionally causes inconvenience, but can be prevented if care be taken to warm the tube to body temperature and having it as clean as possible.

The methods of Roy, Hammerschlag, Haycraft, and others consist in taking two fluids of different densities and noting the specific gravity of a mixture of these two at the point when a drop of blood, on being added, neither sinks nor rises. Different fluids have been used for this purpose, *e.g.* glycerine and water, chloroform and benzol, etc.

These latter methods are apt to give too high results and are troublesome to carry out properly, the exact point at which the specific gravities of the mixture and blood are equal being also very difficult to determine. The volatility of the fluids is also a disturbing factor. Minute details as to the method can be obtained in v. Limbeck's book.

IV. ALKALINITY.—Very numerous methods have been devised to estimate more or less rapidly the alkalinity of the blood. The more exact methods are complex and take much longer time to carry out properly than the clinician has to spare.

The best method to adopt is to estimate the alkalinity of the plasma alone, and at the same time find out how the alkali is distributed in the corpuscles. The first method, which I shall shortly refer to, is that of Haycraft and Williamson, which depends upon the estimation of the alkalinity of the plasma by testing it against a graduated series of glazed red litmus papers. These papers are soaked in different dilutions of normal H_2SO_4, usually about a dozen being required to get exact results. If on taking out a drop of blood and touching one of these graduated papers one get a distinct blue, while on touching one that has been soaked in a slightly stronger solution of acid no blue tint appears (after the corpuscles have been washed off), then the strength of acid required to neutrălise the alkalinity must be represented by an intermediate paper, if there be one. One wishes to find out the exact point where there is neither an excess of alkali nor acid to act on the litmus. There are a number of fallacies in this method; for example, with increased dilution of the plasma, percolation into the litmus papers takes place more easily.

If the total alkalinity of the blood be required, perhaps the most accurate method is that devised by Löwy, only in the majority of cases it cannot be employed with safety, seeing that the patient cannot afford to lose the amount of blood necessary for one estimation. It consists in "laking" a fixed quantity of blood (5 c.c.) with 45 c.c. of a 0·2 per cent ammonium oxalate solution, the alkalinity of a portion of this mixture being then estimated by titration with $\frac{N}{25}$ tartaric acid. Carefully prepared neutral litmus-papers are used as indicators. Other methods, such as that of Landois, are so troublesome to carry out, and are so fallacious, that it is not advisable to give any description of them here.

The distribution of alkali in the corpuscles by micro-chemical methods has been employed by various workers. There is one which can be recommended as giving most interesting results, and that is by the use of erythrosin, a compound which is the same as eosin except that it contains iodine instead of bromine. The precipitate obtained by acidifying the watery solution is easily soluble in ether or chloroform, forming a yellow fluid which, on coming in contact with an alkali, gives a beautiful red colour (formation of a salt). The point of importance in the method is, in the first place, to see that all glass which comes in contact with the blood is purified from salts adhering to it. Then fill a small glass vessel with a chloroform-toluol solution of free erythrosin. Place cover-glass film, face downwards, in this for a minute or two, taking it out as soon as it appears dark red in colour, transfer rapidly to pure chloroform (which is all the better of being changed twice), then mount direct in balsam without drying. The blood picture that is obtained is very instructive, the plasma appearing red, the red cells unstained, the protoplasm of the white corpuscles red and their nuclei unstained. One notices at once the strong alkaline reaction of the lymphocyte protoplasm and the blood plates and the absence of staining in the nuclei of the leucocytes.

V. ESTIMATION OF HÆMOGLOBIN.—Of the methods used for clinical work the following may be regarded as the most suitable, from the point of view of rapidity and ease combined with moderate accuracy:—*Gowers' Hæmoglobinometer, von Fleischl's Hæmometer, Oliver's Hæmoglobinometer.* Of these three the last two are the most accurate, unless great precautions be taken in carrying out the first one. There are two methods more exact than those, which, however, are not so well adapted for clinical work, viz. by Hoppe-Seyler's colorimetric apparatus and Hüfner's spectrophotometer.

Gowers' Hæmoglobinometer.—This method is a colorimetric one and consists in the comparison of a more or less diluted solution of blood against a colour standard which is fixed. This standard consists of a solution of picric acid and picrocarmine in glycerine and water, gelatine being added in order to solidify it. The mixture so obtained is supposed to represent in tint a 1 per cent watery solution of blood. The instrument consists either of two circular glass tubes of the same calibre, or, preferably, tubes which have been flattened in order to obtain a layer uniform in thickness. They may be placed side by side with a slight interval between, or with their edges slightly overlapping, in order to prevent white light passing through between them. The one is the standard, the other a tube graduated from 10 up to 120 degrees of dilution.

Twenty cubic millimetres of blood are measured by means of a capillary pipette, and are then as quickly as possible blown out into the empty tube in which a few drops of water have been previously placed. The tube is quickly shaken in order to defibrinate the blood, and then by means of a pipette stopper, which fits into a bottle containing distilled water, the latter is carefully added drop by drop until the tint in the tube is the same as that of the standard. Complete admixture must be obtained by shaking the tube at intervals during the addition of the water.

The normal blood, on being diluted up to the mark 100, ought to give the same tint as the standard tube, on being examined in the direct line of light. It is better to examine with the tubes in front of a white sheet of paper by daylight or by reflection from a standard artificial light.

The amount of hæmoglobin in the blood is then read off at the point lying between the degree of dilution that gives a slightly darker tint and that giving a slightly fainter tint than the standard. As a rule this instrument is found to be accurate within 4 to 5 per cent.

Von Fleischl's Hæmometer.—In this instrument the blood is diluted to a fixed degree, and the amount of hæmoglobin estimated by comparing the tint with a long wedge of coloured glass, which is of a deep red tint at the thickened end, gradually becoming fainter as it approaches the thin end, where the glass is colourless. This wedge is fixed in a metal frame, which is graduated on one side, so that the percentage of hæmoglobin can be directly read off when the tint of the glass and that of the blood correspond.

The glass wedge can be moved in a horizontal direction, so that any part of it can be brought alongside the compartment of a divided metal cylinder in which the diluted blood is placed. This metal cylinder is fixed on a small stage similar to that of a microscope and is divided into two equal compartments by a partition. At the base it is closed by a glass plate, the wedge being moved horizontally beneath this and viewed by light which has been reflected upwards from a white surface (a reflector covered with calcium sulphate).

The blood is measured by means of a special pipette and then introduced into the proper compartment of the cylinder; distilled water is added and mixed uniformly with the blood. Both compartments are finally filled up with distilled water. Although this instrument is exceedingly handy for rapid estimations, it is not very exact.

Oliver's Hæmoglobinometer.—This instrument is an adaptation of Lovibond's tintometer, which is so largely used at present for estimation of colour intensities of different fluids and solids used in the technical industries. A proper description of this most ingenious instrument and Oliver's modification of it would take up far more space than I have at my disposal, so the reader may be referred to Dr. Oliver's Croonian lectures, where the subject is fully discussed.

It is based upon the same principle as v. Fleischl's instrument, far greater care, however, having been taken in the fixation of the colour standards. The blood, which is taken up by an automatic capillary pipette, is placed in a cell and diluted with water until the mixture is level with the rim. The colour is compared with a series of circular tinted glass standards which have been most accurately graduated. Unfortunately even this method is fallacious, as all must be which are based upon comparisons of tint between coloured glass standards and diluted blood. If the glasses which are sent out with each instrument have not been extremely carefully matched against the original standards (even supposing that the latter are exact) all the precautions which are afterwards adopted to prevent introduction of fallacious readings are thrown away. The nearer one gets to the original hæmoglobin standard the better. Those of us who have worked with the instrument can testify, however, to the rapidity and ease with which, after some experience, hæmoglobin estimations can be made.

Hoppe-Seyler's Colorimetric Method.—This is one of the most accurate methods for estimation of the blood pigment. Here the standard is a solution of carbonic oxide hæmoglobin of fixed strength, and the blood which has to be examined is also saturated with this gas and the tint compared with the normal HbCO solution. If the blood solution be too dark the fluid is run out by means of a tube from the glass chamber in which it is placed and diluted before it is sucked up again, and the tints again compared. The fluid used for dilution is water that has been saturated with carbonic oxide.

Concentrated HbCO solutions in closed capillary tubes can be obtained from the Strassburg Physiological Institute. The glass chamber containing the blood to be examined is fixed in the same frame as that containing the standard HbCO solution. Greater accuracy can be attained by the use o Albrecht's telescopic arrangement with collimator tube.

Estimation of hæmoglobin by the spectrophotometer, although ex tremely exact, is of course impossible in clinical work.

VI. Enumeration of Corpuscles.—The methods employed for thi purpose consist in the estimation of the corpuscles in a fixed amount blood that has been uniformly diluted with a fluid which preserves the cells.

The instrument on the whole most suitable for this purpose is the Thoma-Zeiss hæmocytometer. It consists essentially of a chamber, the depth of which can be fixed, where the cells are counted over squares of known area. A tiny drop of the diluted blood is placed in a glass cell placed on a slide. On the floor of this cell there is a small, circular glass disc, upon the surface of which carefully measured microscopic squares have been ruled. The point of importance is the fixation of the depth of the cell over this disc, with an accurately ground glass gently pressed down over the drop. Newton's colour rings appear, and the depth is then 0·1 mm. The small quadrates are $\frac{1}{400}$ sq. mm. in area, so that the volume of diluted blood over each of these when the cover-glass is applied is $\frac{1}{4000}$ cub. mm. Around each group of sixteen small squares darker lines are drawn, in order to facilitate counting. By a rapid prick with a lancet blood is taken from the lobe of the ear, after due precautions have been taken to ensure cleanliness, and then the point of the carefully dried pipette is inserted into the drop of blood and the latter sucked up to the mark 0·5 c. mm. Any blood adhering to the end of the pipette is removed by the point of the finger cautiously, and then the diluting fluid is drawn up to the mark 101 c. mm., the tube being gently shaken while this is being done. The narrow stem of the pipette is then emptied of diluting fluid by blowing through the upper end, and a tiny drop of the diluted blood in the bulb placed on the centre of the glass cell. Care has to be taken that the drop is not so large that it passes over into the circular valley at the top of the disc on application of the cover-glass. A carefully ground cover-glass is then lowered on the drop, and gentle pressure applied until Newton's rings appear and remain after withdrawal of the pressure. The corpuscles are allowed a few minutes to settle, and then they are counted in as large a number of squares as possible—not less than 48. The average number in each square represents, therefore, the number of red blood corpuscles in $\frac{1}{4000}$ c. mm. of blood diluted 1 in 200, so the calculation of the number in a cubic millimetre of undiluted blood is easily arrived at.

Instead of diluting the blood to this degree, one can get a dilution of 1 in 100 in the bulb by taking blood up to the 1 c. mm. mark, and then drawing up the diluting fluid to 101. Different diluting fluids are employed, as good a one as any being that of Hayem—

Hydrarg. perchlor.	0·5 gms.
Sod. sulphate	5·0 „
Sod. chloride	1·0 „
Aq. dest.	200 c.c.

For the enumeration of the white cells the blood must not be diluted to the same extent, so another pipette which allows of a 1 in 10 or 1 in 20 dilution is employed. The fluid used to dilute is preferably one that will dissolve the reds and at the same time stain the nuclei of the whites. Such a fluid is a ½ per cent watery acetic acid solution, to which a little methyl violet has been added.

In enumeration of the leucocytes, all the squares ought to be counted.

Oliver's apparatus for enumeration of the corpuscles cannot be recommended, as it is by no means so reliable as the Thoma-Zeiss instrument.

B. SPECIAL

I. EXAMINATION OF THE FRESH BLOOD PREPARATION.—A drop of freshly drawn blood is placed on a cover-glass that has been slightly warmed, the latter then being gently lowered on the slide lying on the warm stage of a microscope.

It is well to ring the cover-glass with vaseline. Staining reagents, such as neutral red, may be introduced, and the resultant changes noticed microscopically. These fresh films are mainly of use in allowing one to study the *amœboid activity of the leucocytes*, the *rouleaux formation of the reds*, the *changes in the blood plates*, and the *rate of clotting*.

It is also advisable to examine the blood after diluting with normal saline, or with such preserving fluids as Hayem's or Pacini's solution. All work on dry films should be supplemented by exanination of fresh ones.

II. Fixation and Staining of Blood Films.—Most valuable results have been obtained from the examination of stained blood films. Much of our present knowledge of the different groups of leucocytes and the degenerative changes in the erythrocytes has been obtained from their study.

The first essential is to have suitable cover-glasses which have been properly cleaned. These should be square, not more than 0·1 mm. thick and flexible. They are best cleaned by boiling in a strong H_2SO_4 solution of potassium bichromate (60 grammes potassium bichromate made up to litre with 60 grammes H_2SO_4 in water) for half to one hour, the acid poured off, and the remainder of the fluid removed by first boiling with water and then washing for a long time under the tap. They are then washed with distilled water, and then with absolute alcohol. They must on no account be touched with a cloth after this; if necessary, pass them through the Bunsen flame before use. They are kept in a stoppered glass vessel, and may be picked out with forceps when required. The best forceps to use for the purpose of taking the films are those recommended by Ehrlich—one for fixing the lower cover-glass, and the other, a finely pointed one, for the upper glass. The latter is brought down gently on the top of the drop of blood, care being taken not to get too large a drop, and it is then quickly lowered over the lower cover-glass, which is held by means of the other pair of forceps in the left hand. The blood spreads out evenly, and when this has occurred, the upper glass is quickly drawn across the lower fixed one, care being taken to avoid, as far as possible, pressing or raising the cover-glass that is being moved. The films are then either allowed to dry in the air, or they may be fixed while moist in saturated corrosive sublimate solution, or by some other fixative such as osmic acid. Here I can only shortly run over the principal methods employed for fixation.

The films which have been dried in the air may be fixed by Ehrlich's method, viz. placing the cover-glasses in a drying oven kept at a temperature of 110°-120°. The time during which they are exposed to this temperature varies according to the stain used. With Ehrlich's triacid solution or any of the watery stains only five to ten minutes are required, while for glycerine eosin solutions or for the eosin aurantia and indulin glycerine stain a period of from one to two hours is preferable.

When special attention is going to be paid to the granules, the films ought to be put into the oven at 110°-120° C. from the outset, because one wishes to avoid as far as possible coagulative changes such as occur if the temperature be slowly raised from say 80° up to 120°. It is the rapid removal of water that is important. This method of fixation has certain great advantages. It disturbs as little as possible the chemical characters of the granules, and it preserves the hæmoglobin.

For the finer structure of the nuclei and cytoplasm (spongioplasm) it is not so good as some of the methods to be described later. But in order to obtain a clear blood picture which will afford information as to the different types of leucocytes and the condition of the red cells, this method is essential.

The films which have been dried in the air may be fixed by other agents, *e.g.* equal parts of alcohol and ether for half an hour to two hours, osmic acid or formalin vapour, 10 to 30 per cent alcohol formalin (Gulland), etc.

Fixation by means of alcohol and ether is a good clinical method, and for such a stain as Chenzinsky's methylene blue eosin perhaps the best of all. Osmic acid is good for rapid fixation of the fresh blood for demonstration of blood plates, but it so disturbs the standing properties of the granules as to render it often useless. Formalin vapour and 10 per cent alcoholic formalin solution, as recommended recently by Gulland, are both valuable means of fixation. They act rapidly, are exceedingly good for the demonstration of blood plates, and preserve the contour of the erythrocytes very well. Unfortunately they affect the staining properties of the cells, especially in the case of triacid staining.

The two methods that are most commonly employed for fixation of the moist films are those of Muir and Gulland.

Muir's method.—A few seconds after the films have been taken, they are placed face downwards in saturated $HgCl_2$ solution in 0·75 per cent NaCl for half an hour at a temperature of about 50° C. They are then taken out, washed thoroughly in normal saline, and then taken through 50 per cent alcohol in ·75 per cent NaCl, and the rest of the alcohol series to absolute alcohol. They are then ready for staining with alcoholic stains, or, after washing with water, for treatment with watery stains. This method is of great use for the study of leucocytic structure. It is not good for the study of the erythrocytes, or for the characters of the granules in the leucocytes.

The red cells are frequently very much distorted, and the hæmoglobin is largely removed.

In *Gulland's wet method* the films are left for three to four minutes in the following solution :—

Absol. alcohol saturated with eosin	25 c.c.
Ether	25 c.c.
$HgCl_2$ in absol. alcohol (2 gms. in 10 c.c.)	5 drops

then rapidly washed in water and stained as desired with saturated watery methylene blue, Mayer's hæmatein or triacid, about one minute being sufficient for either of them.

This method is a rapid clinical one, and gives in many cases good results. It also, like the preceding one, distorts the erythrocytes.

As to *the different staining methods* that are employed, there is no limit to their number. The choice will depend upon *what particular element in the film is going to be specially studied.* If it be the specific granules in the cells, then watery alcoholic or glycerine solutions of the principal acid or basic stains are required. If it be for a general examination of all the elements in a film the most useful stains are the triacid, eosin methylene blue and eosin hæmatein mixtures. The first is perhaps the best for the general characters of the granules and for use as a routine stain.

Eosin and methylene blue, or methylene blue (saturated watery solution) alone, is exceedingly good for differentiation of nuclear structure and for basophilous granules. The former gives one of the most beautiful blood pictures that are to be obtained. It is useful also for the study of degenerative changes in the erythrocytes.

Eosin and hæmatein is an exceedingly good stain both for the red and white blood corpuscles. It is also very useful for malarial blood.

There are other methods for the examination of the morphological structure of the corpuscles which are useful for special purposes, *e.g.* fixation of the blood in Flemming's solution, and then taking the corpuscles which have settled, and treating them by the paraffin method. The sections so obtained are treated in the usual way employed for ordinary tissues. In Biondi's *section method* the blood, after fixation in osmic acid, is mixed with agar-agar jelly, then hardened into alcohol, and embedded either in pith or paraffin. The block is then cut in the usual way. The objection to this method is the great dilution of the blood elements.

III. MICRO-CHEMICAL METHODS.—*Glycogen.*—Place the cover-glass film face upwards inside a small bell jar closed tightly below, and having within a few crystals of iodine. The action is finished in a few minutes and the film then mounted in lævulose syrup. The glycogen-holding parts are stained mahogany brown.

Glucose.—Bremer suggested the employment of an eosin-methylene blue mixture for the diagnosis of diabetic blood. He said that the erythrocytes in such a case were not stained with eosin of the reddish violet or red brown tint, as with normal blood, but took on a yellowish green colour, or were unstained. This, however, has been shown now to be by no means a constant change in diabetes, and more or less the same alterations in tint of the red cells are noticed in other conditions.

The same holds of Bremer's more recent methods with Biebrich scarlet and Congo red.

Fat may be present in the form of droplets in the plasma, and also now and then in the leucocytes.

Its character may be detected by its solubility in ether, blackening with 1 per cent osmic acid and resistance to acetic acid.

Pigment granules derived from hæmoglobin may be now and then seen in the plasma, as, for example, in cases of poisoning from sulphonal and potassium chlorate. They may be either crystalline or amorphous, and are distinguished by their brown tint.

Charcot-Leyden crystals vary a good deal in shape, usually colourless rhombic plates or octahedra. They are met with now and then in leucocythæmic blood, and in the sputum. Their source and significance are still unknown.

Spectroscopic Examination.—The spectroscope has not yet been much employed in clinical work, at least in this country. It is, however, frequently important to find out whether an abnormal pigment, such as methæmoglobin, be present or not, as, for example, in poisoning with chlorates of the alkalies. Either a microspectroscope (such as that of Abbé with scale of wave-lengths attached) or a direct vision pocket spectroscope may be employed, the former, however, being the more suitable. It is merely an ordinary microscope, with the spectroscopic part taking the place of the ocular. The little glass cell containing the fluid is focussed first with ordinary eye-piece, and then the spectroscope is substituted and the position of the absorption band or bands noted. I need hardly refer here to the spectrum that one ordinarily gets, viz. oxyhæmoglobin, but emphasise rather the importance of being able to distinguish methæmoglobin. The characteristic absorption band of this pigment lies close to Fraunhofer line C, but on the D side of it. It is a narrow but well-defined band. By action of ammonium sulphide solution this pigment is rapidly reduced to hæmoglobin ("reduced" hæmoglobin).

IV. Bacteriological Examination.—It is necessary at the present day for the clinician to be able to examine the blood in cases of possible organismal infection. Here only a few general directions will be given as to the principal methods employed in bacteriological examinations of blood, as full details will be given in the articles on the different parasites. (See also articles on Septicæmia, Pyæmia, Relapsing Fever, etc.) In order to make this method of examination complete, the blood has to be examined along the following three lines:—

(1) Microscopic examination of films and hanging drop;
(2) Plate and other cultures; and
(3) Inoculations into animals.

Certain precautions have to be adopted in the removal of blood, a good routine plan to adopt being the following:—

Clean the skin over the part from which you wish to get the blood (ear or finger), with nail-brush and warm soap and water, wash with alcohol, then with 1 in 1000 sublimate solution. The latter is then removed by washing with alcohol, and finally with ether. Now constrict the finger with a piece of lint, pass a needle through the Bunsen flame, and rapidly prick the skin. Shake off the first drop, then dip sterilised platinum loop into the fresh drop of blood, avoiding contact with the epidermis, and inoculate successively gelatin and agar tubes. Plate cultures are afterwards to be made.

At the same time films are taken and stained. It is as well to remove the hæmoglobin from the red corpuscles by the following mixture:—

Carbolic acid (5 per cent watery solution) .	6 parts.
NaCl sol. (saturated)	30 „
Glycerin	30 „

A useful stain is that recommended by Vincent, viz. carbol. methylene blue with 1·2 per cent watery gentian violet.

For examination of parasites in living condition, the blood has to be examined in hanging drop on hot stage. Plehn's method for the examination of malaria parasites consists in coating the skin with a thin layer of fluid paraffin, then pricking the skin through this and examining the drop of blood in a layer of fluid paraffin between cover-glass and slide. In many cases it is necessary to get a larger quantity of blood, then one may obtain it directly from a vein on back of hand or the median vein of the forearm. In many cases it is necessary to examine the blood drawn direct from the vein. Of course in all cases the greatest care has to be taken that everything is perfectly sterilised.

A short reference may be made to a reaction that is of great value in the diagnosis of typhoid fever, viz. *Widal's reaction*. It depends upon the fact that the blood serum of typhoid patients from the beginning of the second week of the illness, throughout convalescence, and for a varying period afterwards, possesses (in a dilution of at least 1 in 25) an agglutinating action on typhoid bacilli. If the serum be not diluted so far as this, then the fallacy comes in that blood from persons not suffering from typhoid, in a dilution of at least 1 in 10, may have the same action on the bacilli. This is not to be confused with Pfeiffer's intra-peritoneal reaction, where the immunised serum causes the bacilli to swell up and dissolve. In Widal's reaction the blood is taken from the lobe of the ear, collected in a capillary tube, and is then centrifugalised in order to allow the serum to separate. A platinum loopful of typhoid bouillon culture and one of serum are brought together and examined in hanging drop, or simply spread out on a slide with a cover-glass. If the reaction occurs, then the following dilutions are made in order to determine the point at which agglutination appears (see "Typhoid"):—

·01 c.c. serum in 1 c.c. typhoid bouillon culture = 1 : 100.
·02 ,, ,, ,, ,, ,, = 1 : 50.
·04 ,, ,, ,, ,, ,, = 1 : 25.

The reactions often occur at once, but may be delayed from half an hour to two hours.

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Blood-letting. *See* VENESECTION.

Blood Transfusion. *See* TRANSFUSION.

Boils and Carbuncle.

BOILS

A BOIL is a circumscribed infective inflammation of the skin and subcutaneous tissue, originating either in the pilo-sebaceous follicles or in the sweat-glands, which results in necrosis of the central portion involved, and its subsequent extrusion through a limited opening in the skin as a slough.

Etiology.—Although in all cases a directly exciting cause is necessary, there are nevertheless certain constitutional conditions which tend to favour the development of boils—diabetes, chronic nephritis, chronic alcoholism, and the convalescent stage of the continued fevers. In other words, any condition, physical or hygienic, which tends to reduce the tissue vitality below par, and to lessen the resisting power of the organism as a whole, may be rightly regarded as a predisposing cause. The long-continued application of irritants to the skin may also act locally as a favouring factor; and it is a matter of general observation that the commonest seats of boils are precisely those parts—the nape of the neck, the nates, and the axilla—in which continued friction is most likely to facilitate the entrance of micro-organisms.

Whatever the predisposing cause may be, in all cases we require the presence of the active excitant. This has been proved to be the same organism as that found in all superficial abscesses—the Staphylococcus pyogenes (*S. aureus*, *albus*, and *citreus*). Experimental evidence has been furnished by Garré, who at the same time demonstrated the absolute identity of the organisms concerned in the production of boils and carbuncle. He rubbed vigorously into the hairy, but uninjured, skin of his forearm a pure culture of Staphylococcus pyogenes aureus. Four days later a typical carbuncle had developed in the centre of the inoculated spot, while the periphery was surrounded by a ring of boils of varying sizes. This shows the relation which exists between these two affections and the ducts of the glands, and explains at the same time the occurrence of auto-inoculation. Unna stands almost alone in believing that there is a distinct specific infection for carbuncle, but the researches of Garré, Bockhardt, and others would seem to be conclusive on this point.

Symptoms.—A boil begins as a limited induration of an extremely painful character, the overlying skin being of a vivid red colour. It rapidly becomes acuminated on the surface, and a hair frequently, but not invariably, occupies the centre of the summit. Pain, always severe, varies with the resistance of the overlying tissues; the denser the surrounding structures, the worse the pain—as, for instance, in the perineum, nose, and external auditory meatus. The primary induration gradually widens, forming a definite tumour of varying size, until at the end of from three to six days a tiny pustule develops on the most prominent part, the skin gives way, and a minute quantity of pus is set free. Through this minute opening in the dusky undermined skin a mass of yellowish-gray necrotic tissue can be seen—the "core"—which separates in about a week, leaving a small granulating cavity. This gradually heals up as the subsidence of the inflammatory exudation allows its walls to collapse, and finally leaves a slightly depressed whitish scar. In some cases a boil aborts; the redness, induration, and swelling gradually disappear without suppuration, and the part slowly returns to its normal condition. This, which is often extremely painful, is known as a "blind" boil. Whenever boils occur there is a great tendency towards the occurrence of auto-inoculation—*i.e.* the extension of the suppurative process to neighbouring follicles—and this may occur either as the result of subcutaneous extension from one cellular interspace to another, or more generally by direct infection of the ducts which open on the surface. In this lies the explanation of the frequent phenomenon of multiple satellite boils developing in succession around a central primary focus. Verneuil has described a special form of boil occurring in connection with the coiled portions of the sweat-glands. This is referred to under "Axilla," where it frequently occurs, and the condition has been named "hydradenitis destruens suppurativa."

Treatment.—In the earliest stage, and when a boil is small in size, as in the case of a stye, a cure may often be effected by pulling out the central hair, if present, and touching the spot with nitrate of silver or strong carbolic acid; or if no hair be found, a drop of the latter may be injected into the base of the tumour. Unna strongly recommends the use of a carbol-mercuric plaster-mull, as tending in the early stages to procure abortion, or, in the event of suppuration, to markedly limit its extent and relieve pain. Compresses of glycerine and belladonna, or of saturated solution of camphor in spirit, are also recommended; but in the vast majority of cases early linear incision with a sterilised knife, even before there is distinct evidence of pus-formation, gives the most immediate relief to the pain, and, by reducing tension, limits in a marked degree the extent of tissue necrosis. If the pus has already found vent, the opening should be enlarged, the slough removed, and the cavity lightly packed with antiseptic gauze. Over this hot boric fomentations should be continuously kept. In every case the area surrounding the boil should be well protected by an ointment of salicylic acid and benzoated lard (gr. 30 ad ℥j.), which helps to prevent auto-inoculation with its unpleasant successive crops of boils. When multiple boils appear simultaneously in different regions of the body—a condition described as *furunculosis*—attention must be directed mainly to the patient's hygienic surroundings, and improvement effected in his constitutional state by the judicious use of saline purgatives, by the administration of light, easily assimilable food at frequent intervals, by the exhibition of tonics, and, most important of all, by change to a more invigorating and bracing climate. Specific remedies such as yeast and calcium sulphide are, in the writer's opinion, absolutely worthless.

CARBUNCLE

It will be obvious, from what has been stated above regarding the pathology, that the difference between a boil and a carbuncle is only one of degree; depends, in fact, on the larger amount of specific infection introduced and the wider area of the subcutaneous tissue which is consequently involved. Hence it is attended with greater local destruction of parts and with much graver constitutional disturbance. As a rule, carbuncle attacks persons more advanced in life; those especially in whom the power of general and local resistance is diminished, or in whom the constitutional condition has been lowered either by precedent disease or by unfavourable environment. Its seats of predilection are, in order of frequency, the nape of the neck, the shoulders, face, abdominal wall, and, rarely, the extremities. The most dangerous situations are the scalp and the face, because the greater vascularity of these parts largely increases the dangers of septic absorption, or of septic thrombosis and embolism. The older term "anthrax" should never be applied to this condition, but confined in use to the specific bacillary disease.

Symptoms and course.—A carbuncle begins as a flattened induration of the skin and underlying tissue, which is always accompanied from the first by an exaggerated sensation of tension and by dull throbbing pain in the part. The skin becomes a dusky purplish colour, the pain increases, and the inflammation, unless checked, rapidly infiltrates more and more widely the surrounding tissues. Concomitantly constitutional symptoms develop: great prostration, weakness of pulse, peculiar sallowness of complexion, and, in severer cases, rigors and high temperature. After five or six days vesicles or small pustules form on different parts of the surface, and emit, on breaking, a small quantity of sanious purulent discharge. Through these gradually enlarging openings in the undermined skin may be seen the peculiar ashen-gray sloughs; and, if the case be allowed to run its natural course, these openings coalesce and form a large ragged ulcer, the floor of which is covered with dense adherent portions of necrosed tissue. These slowly separate, and healing takes place by granulation. During this often tedious process the patient may sink from exhaustion, or the graver symptoms of septicæmia may supervene, and rapidly lead to a fatal issue. A form of carbuncle occurring about the lips has been specially described as *malignant facial carbuncle;* but as it differs from the ordinary variety only in the accident of situation and of great local vascularity it does not require separate consideration.

Treatment.—Early and radical treatment offers the best chance of recovery. In the first stages, when the pain is severe, the inflammation spreading, and bogginess has not yet succeeded to brawniness, glycerine and belladonna may be applied underneath hot fomentations As soon, however, as any points of softening can be detected, or any pin-hole openings form, they should be fully incised, the contents thoroughly scraped and cut away, and the exposed tissues freely swabbed with pure carbolic acid. In this way each centre of infection is in turn thoroughly cleansed, free vent is given to the discharge, and the pain is immediately and effectually relieved. The best dressing at first is sterilised muslin wrung out of hot boric solution and frequently renewed. When the sloughs have separated and healthy granulations form, the process of healing will be facilitated by drawing the edges together with circumferential strapping. Besides local measures, the patient's general condition must be carefully watched, and strength maintained by suitable nourishment. Stimulants are generally required, and are best given in the form of egg-flip; and tonics, such as quinine and ammonia, should be regularly administered. Total extirpation of the carbuncle by excision has been practised with successful results; but in milder cases it is not required, and in more extensive cases might possibly add to the gravity of the prognosis. Treatment with anti-streptococcic serum has been tried, but as it is founded on a false pathology it is difficult to see how the improvement claimed can have been otherwise than accidental, except in cases where a general infection has been grafted on a local one. However, it is possible that in the ever-widening development of serum-therapy a reliable remedy for this condition may eventually be found.

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Bone, Diseases of.

THE diseases of bone may be classified as follows:—

1. Bacterial diseases—
    - Pyogenic.
    - Tuberculous.
    - Syphilitic.
2. Parasitic diseases—
    - Actinomycosis.
    - Mycetoma.
    - Hydatid Cysts.
3. Trophic diseases—
    - Including Rickets, Scurvy-Rickets, Osteomalacia, Osteitis deformans, Leontiasis ossia, Fragilitas ossium, and other lesions in diseases of the nervous system.
4. Tumours and Cysts.

The first and fourth of these will be described in the present article. The parasitic and trophic diseases will be considered under their special headings.

INTRODUCTION.—The diseases of bone are under the influence of the same pathological laws as obtain in the diseases of the soft parts. The morbid processes concerned originate and develop in the same way, and lead to the same results as in other tissues. Our conceptions and knowledge of the diseases which may affect the skeleton have been rendered clearer and more accurate by the demonstration of the bacterial origin of the great majority of them, the bacteria concerned being the same as give rise to similar diseases in other parts of the body. The special features of the diseases of bones, as to progress and results, depend very largely on the structural peculiarities of bone as a tissue, and the important changes which take place in the skeleton during the period of growth, and on the frequency with which the bacterial diseases occur in childhood, or at any rate before the growth of the skeleton is completed, thereby entailing functional disturbances of the highest importance. The persistence and tenacity of bone diseases is another of their striking features.

The surgical treatment of diseases of bone has been modified by our conception of their frequent bacterial origin, and our capacity for carrying it out on Listerian principles has enabled us to interfere with a directness and freedom, and at such an early stage, that we are often able to prevent or mitigate the constitutional complications, and the local disastrous consequences which used to be so common, as is evidenced by the older hospital statistics, and by the specimens in museums.

Bacterial Diseases.—It is convenient to include among these the lesions of syphilis and other infective diseases, although the individual organisms concerned have not been satisfactorily demonstrated. Exclusive of the minority, in which the infection of the bone is the result of direct spread from a wound or other adjacent lesion, the diseases in this group result from the infection of the tissues of the bone by organisms carried to the latter through the arteries, and derived either from some pre-existing focus in the body (as is so commonly the case in tubercle), or resulting from the recent entrance of organisms through the skin, or one or other of the internal mucous surfaces. The marrow, especially in the interior of the long bones during the period of active growth, is known to be one of the most favourable of soils for the lodgment of bacteria; the latter, when deposited from the blood in the marrow, may persist in a condition of potential vitality for indefinite periods, long after they have disappeared from the circulating blood from which they were derived. This would appear to be especially true of pyogenic organisms, of tubercle bacilli, and probably also of the infective agent of syphilis. The determination of disease in the bone, resulting from the bacterial lodgment in the marrow, will depend, both as to time of onset, severity and extent, on the initial virulence and number of the organisms, and upon the resistance of the tissues of the individual concerned, and upon the assistance of certain external conditions, such as trauma, strain, exposure to cold, which will act also as localising factors in determining the seat and gravity of the lesion.

The bacterial diseases of bone include all the common acute and chronic affections to which the skeleton is liable (excepting new growths), and are therefore also the cause of the common sequelæ of disease in bone, familiarly known as caries and necrosis.

Pyogenic Diseases of Bone.—These are the result of infection with pyogenic organisms. They are especially met with from birth up till the time the growth of the skeleton is completed. They are predisposed to by exaggerated physiological activity at certain parts of the skeleton. They include not only all the conditions which are more obviously associated with the presence of pus organisms, *e.g.* acute suppurative osteomyelitis, periostitis, epiphysitis, and chronic abscess of bone, but also those which may run their course without the formation of pus, namely, those known as growth-fever, hyperostosis and sclerosis, quiet necrosis, serous or albuminous forms of periostitis and osteomyelitis, certain bone cysts, and probably also the inflammations of bone associated with acute rheumatism. It was formerly assumed that pyogenic organisms were only concerned in lesions of the bones which were attended with the formation of pus, and especially with those which ran an acute or subacute course. They include, however, every variety of lesion from congestion to suppuration. Although this knowledge of the wider influence of pyogenic organisms in the production of bone disease has simplified things from the pathological standpoint, it may be the source of greater difficulty in their clinical recognition, because the exact bacterial nature of many of the more chronic forms of bone disease may not be recognised unless the infected tissues are submitted to special bacteriological investigation. Still, if such clinical recognition is not always possible, it is better on the whole to accept the difficulty or impossibility than to make the mistake which must have frequently been committed, of describing chronic bone lesions as of tuberculous or syphilitic origin, when in reality they are the result of infection with pyogenic organisms. Inasmuch as the vast majority of pyogenic diseases of bone are the result of infection with the Staphylococcus aureus, these will be described in the first instance; those due to other pyogenic organisms will be dealt with later.

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I. DISEASES OF BONE DUE TO THE STAPHYLOCOCCUS AUREUS

Staphylococcal Osteomyelitis and Periostitis.—The bone marrow, during the period of growth of the skeleton, constitutes a specially favourable seat for the decomposition and development of staphylococci. They are mainly transferred from one point of the body to another by leucocytes. The site of entrance and method of infection of the staphylococci are not always apparent. This is especially the case in the more severe forms of the disease, which appear to originate spontaneously with rapid onset of general illness. In these there is probably a high degree of initial virulence on the part of the cocci, and their mere entrance into the body and into the bones may suffice to produce disease without any accessory or localising factors.

The site of entrance is frequently the skin: the outbreak of the disease is often preceded by eczema, acne pustules, blisters, small infected wounds, vaccination sores, impetigo, and by septic conditions at the umbilicus in the newly-born. Kocher believes the cocci first acquire a higher degree of virulence in the skin of the patient, which acts as a nursing-ground for their further development. The internal mucous surfaces may be the site of entrance; the proof of this is obtained with greater difficulty; it may be assumed, however, from osteomyelitis being preceded by aphthous stomatitis, teething, foul furred tongue, excoriations and fissures in tongue, mouth, nose, and by adenoids, by small tonsillar abscesses, and by intestinal disturbances, such as diarrhœa. It must be assumed that staphylococci may enter the general circulation and be deposited in the living state in the marrow without there being any external evidences of such infection, or only such minor disturbances of health as may be shown by slight indisposition, or feverishness, and shooting rheumatic pains in the limbs.

The localisation and successful development of the infection in the bones may be assisted or determined by injury (blow, squeeze, or twist), by exertion (a long walk), or by exposure to cold (wading), all of which act by causing extravasation of blood or disturbance of the circulation and nutrition. Volkmann has described cases in which years have elapsed after recovery from osteomyelitis, and yet a blow on the affected bone has been followed by sudden violent inflammation. The injury may, on the other hand, precede the infection by weeks or months; the outbreak then occurs at the injured area, when some skin infection (*e.g.* boils) affords a means of entrance for the staphylococci. The reverse is observed when a pustular affection of the skin precedes the trauma; the former may be healed or cured when some accidental injury allows of the development of the cocci which, up till then, had been lying dormant in the marrow. The trauma and the infection may be simultaneous, as when a patient with cutaneous boils falls on his knees and develops acute osteomyelitis (Kocher).

One must assume that staphylococci may persist in a condition of potential vitality for months or years in the bone marrow, otherwise the outbreak of acute osteomyelitis after an injury would be impossible of explanation. They are still more capable of lying latent in diseased tissues, witness the breaking out afresh, it may be on one or upon several occasions, of old and apparently cured osteomyelitis after the lapse of many years. The less severe or chronic forms of osteomyelitis are by no means so exceptional as was previously believed. Their occurrence is to be explained either by a greater resisting power on the part of the tissue, whereby the conditions for the development of the cocci are most unfavourable, or by assuming the possibility of infection by an attenuated virus.

1. ACUTE SUPPURATIVE OSTEOMYELITIS AND PERIOSTITIS

Definition.
Bones affected.
Influence of age, sex, and constitution.
Pathological anatomy.
  1. IN THE LONG BONES DURING THE PERIOD OF GROWTH.
  2. IN THE LONG BONES OF YOUNG INFANTS.
  3. IN THE LONG BONES OF ADULTS.
  4. IN THE SHORT AND FLAT BONES.

Clinical features.
  1. IN AVERAGE OR TYPICAL CASES.
  2. IN GRAVER CASES ALLIED TO PYÆMIA.

Sequelæ.
Diagnosis.
Prognosis.
Treatment.

Acute Suppurative Osteomyelitis and Periostitis.—Synonyms—Typhus of the extremities, acute necrosis, juxta-epiphysial osteomyelitis, acute epiphysitis.

Definition.—A staphylococcal inflammation of the marrow and periosteum in certain predisposed parts of the skeleton, observed in childhood and youth, often grave in character; attended with the formation of pus, with damage to the bone-forming tissues, and frequently resulting in death of bone.

Bones affected.—The long tubular bones take precedence in the following order of frequency:—1. Lower end of femur. Either end of tibia. 2. Upper end of humerus. 3. Ulna, fibula, radius. 4. Metatarsals, metacarpals, phalanges. *The other bones* of the skeleton are occasionally affected.

Liability according to age, sex, and constitution.

Age.—10-14 and 13-17 have been stated as maxima; the disease is very rare after 20, when the skeleton has reached maturity.

Sex.—Males are affected more often than females in the proportion of 3 to 1, because they are more exposed to injury, to violent exertion, to strain, and to cold.

Constitution.—The disease does not necessarily pick out the weakly, but may affect the most robust, whether from country or town. It is not uncommon after other infective diseases, *e.g.* scarlet, small-pox, typhoid.

During the period of growth, primary inflammation in the marrow predominates; periostitis is usually a secondary lesion. This fact must be emphasised from the outset, because the clinical features are misleading, inasmuch as the periosteal element often appears in the foreground. As a rule the disease commences and is most intense in the ossifying junction at one end of the bone. Rarely it commences at both ends simultaneously (bipolar osteomyelitis), or it may commence at one end and spread to the other. Osteomyelitis in the marrow of the epiphysis itself (acute epiphysitis) is very rare as an independent lesion.

Pathological Anatomy.—The changes observed vary with the type of bone affected, whether a long bone or a flat one, and with its age, whether before or after maturity.

1. IN A LONG BONE DURING THE PERIOD OF GROWTH the changes in the tissues affected are those of intense inflammation, gradually merging into those of suppuration. The later appearances partake rather of purulent infiltration than of actual collections of pus. Where the process is most advanced, *i.e.* at the ossifying junction, there will be evidences of absorption, rarefaction, and disappearance of the framework of the bone, and of the semi-calcified tissue in process of conversion into bone, enlargement of the marrow spaces and of the Haversian canals, the formation of visible tunnels and of spaces, occupied by greenish yellow pus with corresponding destruction of the soft tissues, and therefore of the bone-forming elements. The formation of spaces and tunnels by rarefaction and absorption is always most marked in the region of the ossifying junction, the larger spaces are near the epiphysial disc itself, and they often communicate with the exterior through holes in the cortex. The blood-vessels within the marrow undergo obliteration to a considerable extent, and the veins become the seat of thrombosis and of infective phlebitis.

In addition to the changes in the framework of the bone, to which reference has been made, there frequently occurs *the visible death or necrosis* of considerable portions, or of the entire bone, in the region affected. The death of the bone is not to be attributed to vascular obliteration or to tension, or to deprivation or destruction of its soft parts, but to the poisonous action of the bacterial products in the pus, with which it is in contact. Doubtless the vitality of the bone is seriously impaired by the vascular and tissue changes which attend the process, but the actual death is the result of intoxication. It is no longer held that bone, bared of its periosteum, or deprived of its marrow, necessarily dies. The occurrence of aseptic necrosis is no longer admitted. This is borne out by the effects of the Listerian treatment of osteomyelitis, it being found that necrosis is a much less frequent result than in older days, in which septic infection was allowed to take place from without, through the incision, or through sinuses opening on the surface.

To return to the inflammation and suppuration at the ossifying junction, it may remain localised to the latter region, or it may spread along the medullary canal for a varying distance, or throughout its whole extent, or it may extend through the shell or cortex of the bone overlying the ossifying junction in which it originated, to the deeper surface of the periosteum, by way of the Haversian canals, or through an enlargement of one or more of these, amounting to a visible perforation of the bone; it then spreads along the deeper layer of the periosteum, and presents the features of *periosteal inflammation and suppuration*. The extent of the latter corresponds generally to the extent of the lesion in the marrow. The pus accumulates under the periosteum, and lifts it up from the bone. In the region of the ossifying junction, where the pus first erupts through the bone, it lifts up the periosteum with difficulty because of its intimate connection with the bone; extension along the shaft is easier than towards the epiphysis and joint, which accounts for the more extensive stripping of the shaft, it may be in its whole circumference, or at points of special attachment to the bone these may constitute a barrier. It may, however, extend continuously throughout the entire shaft. Less frequently there may be several distinct abscesses under the periosteum, scattered up and down the shaft, each being derived from a focus of suppuration in the subjacent marrow. The sub-periosteal pus may be observed to communicate, as described, with that in the marrow, and also with collections of pus in the overlying cellular tissue. When there is pus in the marrow there is always pus under the periosteum, and the converse is usually true. The sub-periosteal pus makes its way to the surface by the easiest anatomical route, erupts through the skin, and discharges externally. The discharging canal is known as a sinus. Along with, or apart from, the spread from the marrow to the periosteum, the spread may tend in the direction of the adjacent joint, by extension under the periosteum and capsule, or through the epiphysis and articular cartilage, or from the periphery of the epiphysial junction, especially if the latter is entirely intra-articular, as is the case in the hip.

The occurrence of purely periosteal suppuration is admitted by certain authors; it is probably the exception in young subjects; when it does occur, and the pus is evacuated, the periosteum comes again into contact with the bone, and recovery is more likely to take place without necrosis.

From absorption and softening of bone at the juxta-epiphysiary region in severe forms of inflammation, *separation or displacement or bending may take place through the junction*, and on the diaphysial side of the epiphysis, the epiphysial disc and the epiphysis itself being intact.

The epiphysial cartilage may remain intact, or it may suffer from the infection. It may be partially destroyed by perforations or tunnels through its substance, or it may be completely destroyed, while at the same time the periosteum surrounding the epiphysis may be lifted up by pus. Where it is completely destroyed, the bony surfaces of the diaphysis and of the epiphysis are opposed to each other by irregular eroded surfaces bathed with pus, and are only held together by periosteal attachments, or by the remains of these at the periphery.

Displacement or separation of the epiphysis (epiphysiolysis) may then take place by muscular action or some movement of the limb. The destruction of the epiphysial disc may entail partial or more or less complete loss of its bone-forming functions in the future, according to the physiological importance of the epiphysial disc concerned, and consequent dwarfing of the bone. There are exceptional cases in which destruction of the disc at one end of a bone has not interfered with its symmetrical growth. Concerning *necrosis in the epiphysis itself*, it is usually partial in the form of small, central, spongy sequestra; the entire epiphysis very rarely dies, and separates as a sequestrum, *e.g.* head of femur.

The adjacent joint may be influenced by the mere vicinity of the inflammation in the adjacent bone, in which case it fills with a serous effusion similar to that causing œdema in the soft parts. This effusion has been found sterile on examination. *When the joint is actually infected* the lesion assumes the characters of *an infective arthritis*, which, from its frequency during the earlier years of life, has been called *the acute arthritis of infants*.

Repair.—*Separation of the dead bone, and formation of new bone in its place.*—When the acute stage of the process has subsided, a process of repair sets in; the latter goes on often under the most unfavourable conditions, in the shape of persistent suppuration and depraved general health. The sub-periosteal and medullary marrow, wherever it has retained its vitality, resumes its osteogenetic functions, often in an exaggerated or exalted manner. The initial changes in this direction consist, as in the repair of lesions in other tissues, in the transformation of the living periosteum and marrow into granulation tissue.

Wherever the periosteum has been lifted up from the shaft by an accumulation of pus, or is in contact with bone which is dead, it proceeds to form new bone with great activity; the new bone thus formed is distinct from that which has perished; hence, in the course of time the dead shaft becomes invested or surrounded by a *tube, sheath, case, or involucrum of new bone*, more or less complete, according to the integrity of the periosteum and extent of the necrosis. Wherever the periosteum has been perforated by the pus making its way to the skin surface, there will be defects or holes in the involucrum, which are called *cloacæ;* they correspond, more or less, in position to the sinuses in the skin; hence in passing a probe down one of the sinuses, it usually passes through the corresponding cloaca, and strikes the dead bone lying in the interior of the involucrum. Where the periosteum has been more extensively destroyed there will be corresponding gaps or defects in the new case, or new bone may only be formed in small patches here and there, or no new bone may be formed at all. The tissue cells and capillary loops on the internal aspect of the new case acquire the appearance of a lining of granulations, which, if in immediate contact with the dead bone beneath, will eat into the surface, and partly absorb it; the dead bone in the process of separation may thus acquire a pitted, grooved, or worm-eaten appearance, and may undergo diminution in size. If the dead bone is merely in contact with pus, its surface remains smooth and intact. The separation of the dead bone from the living takes place at the line of junction between the two, and is achieved by the activity of the cells of the marrow, chiefly osteoclasts, which eat both into the living and into the dead bone, so that separation takes place at the expense of both; ultimately *the dead bone becomes loose*, and in the macerated specimen will rattle about in its new case. The loose separated dead bone is called a *sequestrum*, and the latter presents on the surfaces and edges, with which it was originally continuous with the living, an eroded or worm-eaten appearance. The worm-eaten appearance of sequestra is produced at the moment of their separation, and not afterwards.

In sequestra derived from the diaphysis one end frequently corresponds to the ossifying junction where the separation has taken place transversely. The separation of sequestra is more rapid at the spongy end of the shaft than towards its centre, where the line of separation is often oblique.

Usually the sequestrum is dead white in colour, but it may be discoloured by the products of decomposition. Sequestra are described as total, partial, superficial, central, cylindrical, tubular, annular, according to the extent, position, and shape of the bone which has perished.

If the necrosis is central, and does not involve the surface bone, the latter becomes vascularised and canalised by cells and vessels which have to do with the separation of the dead bone beneath, while on its periosteal aspect it contributes, along with the periosteum, to the formation of the new case. Where the foci of suppuration have been scattered up and down the medullary cavity, and the bone has died in patches, several sequestra may be included by the new case; the latter is then incomplete, and will be continuous with, and fused here and there with those parts of the original shaft which have not died. Each patch or portion of dead bone is slowly separated, and comes to lie in a cavity lined by granulations, the sequestrum being again smaller than the portion of living bone which it represents.

As a result of the participation of the marrow in the formation of new bone, the medullary canal may be obliterated at one or more points, or throughout its extent, resulting in great increase in the weight and density (sclerosis) of the bone.

The new case is at first spongy and fragile, but in the course of time it becomes denser and stronger. Also, wherever the old bone has been rarefied and vascularised, there is subsequent condensation and hyperostosis, often going beyond the normal. Wherever the periosteum has not been detached, the new bone which it forms is deposited on the original shaft in a gradually-diminishing amount, according to the distance from the seat of infection. It results in increase in the girth of the bone (hyperostosis).

Spontaneous Fracture.—The juxta-epiphysiary form has already been referred to. Spontaneous fracture of the shaft proper may also occur before repair is complete (towards the end of sixth week), from standing on the limb, or by the action of muscles. It occurs at the site of necrosis, or where the bone is mined by rarefying osteitis, and is usually met with in the femur in its upper or lower third. The fracture may involve both the old bone and the new case, or one or other of these alone. Short of fracture, there may be bending or curving of the shaft from want of rigidity; this especially affects the new case, resulting in great deformity and shortening of the limb.

The Spontaneous Extrusion of a Sequestrum may occur from the pressure of granulations upon it, provided there be a cloaca large enough (or sequestrum small enough), and the cloaca is so situated as to favour its escape externally. The surgeon has usually to interfere and perform the operation of sequestrotomy. The displacement or partial extrusion of a sequestrum may be the cause of serious complications, *e.g.* a sequestrum in the position of the trigone of the femur may perforate the popliteal artery or the cavity of the knee-joint.

The capacity for the reproduction of bone which has been lost varies.—Certain portions of bone normally covered by cartilage (*e.g.* head and neck of femur, astragalus), and bones which are entirely formed in membrane (flat bones of skull, upper jaw), are not reproduced.

Certain differences are observed when the disease affects *the long bones of young infants* before the epiphyses are properly constituted; the epiphysial cartilage is not as yet differentiated from the articular cartilage, and is in immediate relation with the capsule of the joint, in other words, it is intra-articular. Juxta-epiphysiary osteomyelitis is therefore the prevailing lesion in young infants, and not epiphysitis, and the infection is much more likely to involve the adjacent joint than to spread to the diaphysis and cause necrosis, as in older children.

In the long bone of an adult, after maturity has been reached, the anatomical arrangement is not only very different, but the special predisposition of the marrow is lost. More favourable conditions are required in order that the cocci may flourish and produce disease. Acute osteomyelitis is very rare. There is greater liability to chronic forms of disease, either starting as such, or met with as relapses of an antecedent acute infection.

In short and flat bones, when these possess an ossifying junction, as in the os calcis, the lesion is similar to that in the long bones. *In small bones generally* the lesion tends to assume the characters of a joint disease. *In flat bones*, such as those of the skull, ilium, periosteal suppuration is the predominating feature, and suppuration may occur on one or on both aspects of the bone. In the skull the infection may spread to the membranes and to the venous sinuses. Necrosis may occur rapidly. The dead bone in the case of the skull is very slow in separating. In the skull, upper jaw, malar bone, and nose there is no new formation of bone to replace that which has been lost.

Clinical Features.—In a general survey of these, it is to be borne in mind that there are two sets of symptoms: the *local symptoms, i.e.* those resulting from the localisation of the infection in the bone or bones, and the *general or constitutional symptoms*, resulting from the diffusion of the staphylococci or their products throughout the system by means of the circulation. The latter may be further differentiated into (1) *primary constitutional symptoms*, referable to their diffusion throughout the system prior to their localisation in the bones; and (2) *secondary constitutional symptoms*, referable to the absorption of toxines, or to general re-infection from the local lesion in the bones, and succeeding the latter. Of these the second are always present in some degree; while the first may be absent, or not recognised, or only indicated in the previous history of the patient, *i.e.* in those cases in which the cocci have already effected a lodgment in the bone marrow, and are lying latent or dormant, and are only waiting the help of some accessory factor (*e.g.* blow, twist, exposure to cold), in order to blossom out and produce their toxic effects. In these cases the local symptoms will play the leading part in the clinical features, and will only be followed later, or after an interval, by the secondary constitutional symptoms. Where the systemic infection is recent and virulent, then we have primary constitutional symptoms, such as delirium and prostration, before there are any evidences of localisation in the bones; in fact the patient may actually succumb before these latter manifest themselves at all, the death being due to general staphylococcal infection. On the other hand, the local symptoms may precede the general, and may be the most prominent features of the disease; these are the milder, less fatal forms. All intermediate forms between these extremes are met with. It is therefore very difficult to give a description of the clinical features which will embrace all the clinical variations.

Local Symptoms.—The onset of the disease *IN AVERAGE TYPICAL CASES* is characterised by local symptoms—(1) *pain*, which may develop suddenly and violently, or may gradually increase from day to day; it is often assumed to be traumatic or rheumatic in nature. The initial seat of the disease being so commonly at the joint end of a long bone, there is early and pronounced *disturbance of function* in bearing the weight on the limb or in movement, frequently suggesting a joint lesion, for which treatment is instituted and time lost. (2) *Tenderness on pressure* is of great value in recognising the osseous seat of the lesion and the part of the bone affected, also pain on attempted manipulation, as if the limb were fractured.

Sooner or later there are *constitutional symptoms*. The earlier and more prominent the febrile symptoms, the more acute and serious the disease. The temperature runs up with a rigor to 102°-104° F., with general illness, headache, loss of appetite, vomiting, and it may be convulsions in young children, rapid pulse, and pains in the limbs other than the one specially affected. The temperature usually mounts from 101°-102° in the morning to 103°-104° in the evening, but it runs a very variable course.

In the course of time there develop *characteristic local signs swelling* in the region of the ossifying junction, *œdema* of the soft parts, and *dilatation of the surface plexus of veins*. The appearance of the swelling is comparatively late in the majority of cases, because of the predominance of osteomyelitis as the primary lesion. The date of its appearance depend further on the depth from the surface of the bone affected; it appear earlier in the tibia, ulna, or clavicle, than in the femur or humerus. Th swelling may be less evident to the eye than to the fingers on carefu palpation (by one suspecting the disease and looking for it). Th maximum swelling or thickening of the bone, and the tenderness, correspon to the junction of the shaft with the epiphysis, stopping abruptly at th level of the epiphysial disc, and tailing off gradually towards the diaphysi or it may spread along the diaphysis. It is best appreciated by palpatin the bone from the middle of its shaft towards its end. As time goes o there may be *diffuse redness* of the skin in the case of superficial bones, suc as the tibia. The swelling over its most prominent part becomes elastic an more yielding, and finally yields definite evidences of *fluctuation*, indicati of the elevation of the periosteum, and of the accumulation of pus benea it. This stage may be reached at the end of twenty-four hours, or not f several (eight to fourteen) days. When the bone is deeply seated (upper e of the femur or humerus), the swelling develops late and is ill-defined; t surface skin may present no alteration whatever, or only a dilatation of t superficial plexus of veins; on palpation the soft parts may present a feeli of resistance.

Fluctuation finally reaches the surface, until, either with the help of t knife, or spontaneously, at the end of ten days or a fortnight, the swelli bursts, and a large quantity of matter escapes, mixed or not with blo often with considerable amount of fat droplets, and, it may be, min particles of bone. These latter confirm the medullary origin of the suppuration. The fever then remits, and the pains and other symptoms are relieved. In acute cases in which fluctuation is apparent, one finds on incision that the periosteum is lifted up, and that the bone is bare; the extent of this corresponds fairly accurately with the extent of the lesion in the marrow. Holes may or may not be seen in the cortex. According to the situation, *i.e.* the relation of the epiphysial junction to the synovial cavity, one may find the adjacent joint infected or not.

If the periosteum be extensively lifted up, one may assume the probability of necrosis involving more or less of the diaphysis, especially if infection has been allowed to take place from the skin surface. Such infection reinforces that which exists already in the interior of the bone, and which may have become worn out or attenuated. The entrance of other organisms complicates the general intoxication, and renders it more serious. In less severe cases, and especially in the minority, where the suppuration is purely periosteal, healing may occur without necrosis. This healing, however, may be only temporary; at any subsequent period of life the infection may break out again with the formation of pus, the reopening of the old scar, and, it may be, the formation of a sequestrum (relapsing osteomyelitis).

LOCAL COMPLICATIONS.—The *symptoms of involvement of the adjacent joint* vary from those of simple effusion to those of acute arthritis. The latter may account for little in the clinical picture, being merely an addition to, or an extension of, the suppuration in the bone, or the arthritis may predominate, and its symptoms overshadow those of the bone suppuration from which it took its origin; in the latter cases the bone lesion is apt to be overlooked.

Separation through the ossifying junction between the disc and the diaphysis may occur insidiously between two dressings. It reveals itself by an alteration in the attitude of the limb; the direction of the displacement is influenced by gravity or by the action of muscles.

It may resemble a dislocation, but may be differentiated by making out the exact seat of the abnormal mobility, and by recognising dry grating or crepitus.

Separation of the Epiphysis itself.—This is alleged to be rarer than the above, except the epiphysis at the upper end of the femur; partial displacement or gliding of the epiphysis is common in other situations. It is often accompanied by suppuration in the joint. It may take place suddenly or gradually, and may occur without suppuration. Epiphyses which have separated may die and be extruded, or reabsorbed, or they may reunite.

Spontaneous fracture of the shaft has chiefly been observed in the femur. It may take place after some effort or slight injury; it is attended by the usual symptoms of fracture. It may unite quite satisfactorily under treatment.

Spontaneous dislocation has been chiefly observed in the hip-joint; it may be attended or preceded by suppuration in the joint, or there may be no suppuration. The hip may dislocate without suppuration, while at the same time the lesion in the shaft of the femur may suppurate in the ordinary way, and lead to necrosis.

Special Clinical Features in Skull and Pelvis.—In the skull the disease is fortunately very rare, and is chiefly met with in young children. There is intense pain in the head, the child is somnolent and drowsy, and this condition may pass into delirium and coma if left alone. Swelling may be late; necrosis takes place early. The lesion may be complicated by meningitis or sinus phlebitis, and is then usually fatal.

In the pelvis the disease is usually grave in character, and often terminates fatally.

In more serious cases, in contrast to what has been described in average typical cases, we have a condition related to what is generally meant by the term "pyæmia" in its acute and chronic forms (staphylo-hæmia, Kocher). There are cases in which *the lesions in the bones and joints are multiple,* but in which the initial lesion in point of priority and intensity stands out prominently in the clinical picture. The secondary or metastatic lesions in the skeleton, proceeding from the initial focus, may follow the same clinical course as the original one, or they may run a milder course; they may involute and subside altogether, or result in thickening of the bone or apparently resolve only to develop at a later period into an abscess of the bone.

The influence of these secondary lesions on the primary one is injurious—the primary one is more liable to progress without limitation; the toxins produced in the other lesions depress the resisting power of the tissues, and give the staphylococci a freer hand. The result frequently is that the patient dies of intoxication. The toxic disturbance of the liver and digestive organs accounts for the loss of appetite, the dry tongue, the distension of the abdomen, the constipation and the intercurrent diarrhœa, decomposition of the intestinal contents, and auto-intoxication; the parenchymatous degeneration of the heart muscle leads to cardiac weakness, and a small, rapid pulse.

The kidney frequently suffers from bacterial and from toxic nephritis; the urine is usually scanty and contains albumen. The skin may show erythematous and purpuric rashes of a red or claret colour; there may be raised eruptions like urticaria, and others suggesting some eruptive fever. The fever corresponds to the absorption of toxins; it is remittent and very irregular; the maximum may be in the morning in the shape of an abrupt and rapid rise from the normal. All these intoxications are aggravated by fresh infection from without at the time of spontaneous rupture of the suppuration on the skin surface, or where the suppuration has been opened without antiseptic precautions.

In the same way that the primary lesion may cause death by metastasis and general intoxication, the secondary ones may come into prominence, and may contribute directly to a fatal result. Unless great care is taken secondary foci may remain undiscovered, not only in the bones, but in the joints, and especially in those which are deeply seated (shoulder and hip) Similarly periosteal and joint suppurations may erupt into the surrounding tissues, and lead to large inter- and intra-muscular collections of pus, which cannot be recognised and dealt with early enough.

Similar intoxications may be due to *metastases in the internal organs* which are difficult to diagnose and to evacuate. The more numerous th secondary foci, the greater is the risk of too late or ineffectual removal o toxines from the body. The most important of these internal metastase are met with in the lungs and kidneys, *e.g.* abscess of the lung, pleurisy empyema (for which incision and drainage are required with or withou rib-resection), pyonephrosis, abscess in the parotid, in the liver, and othe organs.

Endocarditis with widespread infective embolism; thrombosis an phlebitis in periosteal, subcutaneous, or larger veins from staphylococcu invasion of their walls, are to be regarded as complications of exception gravity.

In these severe cases with internal metastases the *clinical features* approach the typhoid state.

The temperature is persistently or intermittently high; the pulse is small and rapid; the patient is breathless; the senses are blunted; there are cramps, as in meningitis; the lips and tongue are dry; the gums swollen and covered with sordes; the belly is swollen and sensitive; the spleen is swollen; there may be diarrhœa and vomiting; and there may be albumen in the urine. Staphylococci may be cultivated direct from the living blood. The picture of pyæmia may be complete. These grave cases may begin in the same way as a medullary suppuration confined to one bone, or the disease may be multiple, metastatic, and pyæmic from the onset. All grades are found between localised osteomyelitis and pyæmia.

THE RESULTS OF ACUTE SUPPURATIVE OSTEOMYELITIS.—Apart from the relapsing and chronic forms of osteomyelitis following on the acute attack which are considered later, it will be of service to enumerate the results or sequelæ of one attack of acute suppurative osteomyelitis, not only those immediately following the attack, *e.g.* necrosis, but those which may not become evident till a considerable time has elapsed. These latter refer especially to the influences of the attack on the future growth of the bone and the production of deformities.

Persistent Sinus or Sinuses.—These usually indicate the presence of a sequestrum. The patient may not apply for treatment until the attack has passed, and he comes complaining of these sinuses.

As compared with tuberculous sinuses, those resulting from suppurative osteomyelitis are depressed, adherent, with rigid edges; the surrounding tissues are dry and hard, and the bones are markedly thickened; whereas in tuberculous disease of bone the sinuses present pouting or fungating granulations, the edges of the sinus are undermined, and one may squeeze from them caseous debris and thin watery pus; thickening of the bone is usually much less pronounced.

The sinus may migrate in the course of years as a result of the growth at the epiphysial junction, and may come to lie higher up the shaft than it did originally. Displacement of the sequestrum may result in penetration of the adjacent joint, or injury of the popliteal or other artery.

In the pelvis, the sequestrum may make its way into the bladder.

The sinus may become the seat of cancer (squamous epithelioma), if it be allowed to persist for years—this has been especially observed in sinuses over the tibia, the femur, and the os calcis. The edges of the sinus become indurated and the seat of a warty or cauliflower-like growth, which also penetrates into the bone; the sinus is converted into an open excavated ulcer with stinking discharge.

Deformities resulting from Acute Osteomyelitis.—These will be the more marked the earlier in life the attack occurred.

Shortening of bone is fairly common from interference or arrest of growth, resulting from osteomyelitis at the ossifying junction, and of partial or complete destruction of the ossifying tissues. The loss in length corresponds to the extent of the destruction and to the functional value of the epiphysial junction concerned. The shortening may not only affect the bone originally the seat of the osteomyelitis, but may affect the other bones of the limb, so that the whole limb may be notably shortened.

Lengthening of bone, from exaggerated growth at the epiphysial junction, may occur when the latter escapes the direct injurious effects of osteomyelitis, *i.e.* where the disease has involved the shaft of a long bone. The increase in length may be accompanied by increase in girth and density (hyperostosis and sclerosis). It is much rarer to have exaggerated growth in the length of a bone adjacent to the one affected, *e.g.* the femur in disease of the tibia. In segments of limbs where there are two parallel bones, the arrest of growth or the exaggerated growth of either results in curvature of the bone which is too long, and in alteration of its articular surfaces.

Twisting or curvature of a single bone, *e.g.* femur or humerus, from bending of the new periosteal bone after removal of the dead diaphysis, is associated with very evident deformity, consisting in shortening and alteration of the axis of the limb.

There may result an obliquity of the bone from unequal destruction of the epiphysial cartilage, where one half is destroyed, and the other half goes on growing, *e.g.* genu valgum, cubitus valgus, club hand.

A spontaneous fracture of the femur, tibia or humerus may have been allowed to unite with deformity.

Deformity may result from permanent displacement of an epiphysis. Such deformities are more often observed in relation to the lower end of the femur, or the upper end of the tibia. They resemble incomplete dislocations.

Joints may become contracted or ankylosed. The latter may be fibrous or osseous, and in good or in bad position. Joints may also be partially or completely dislocated.

Combinations of the above deformities in a limb render it unsightly and useless. Extraordinary examples may be met with in neglected cases.

THE DIAGNOSIS OF ACUTE STAPHYLOCOCCUS OSTEOMYELITIS AND PERIOSTITIS IS TO BE MADE—1. *From Erysipelas, Cellutitis, and other inflammations of the overlying soft parts,* chiefly confusing in superficial bones, such as the tibia, clavicle or ulna. The features of inflammation and of suppuration appear early, are superficial and more evident lesions from the first than those proceeding from disease of the subjacent bone. Careful search should be made for tenderness in the position of the ossifying junction. *Erythema nodosum* has been mistaken for osteomyelitis of the tibia.

2. From sub-periosteal hæmorrhage in the shaft of a long bone, in hæmophilia and in scurvy-rickets.

3. *From the Eruptive Fevers.*—One is most likely to confuse the more severe forms of osteomyelitis when there is early and pronounced general intoxication with certain of the malignant fevers. In all doubtful feverish conditions in children and in young adults, the ossifying junctions of the long bones should be systematically searched for areas of pain and tenderness.

4. *From Acute Articular Rheumatism* (rheumatic fever).—Kocher appears to regard the osteomyelitis of young individuals as the same disease as acute articular rheumatism. Professor Sahli of Bern found the same staphylococcus in a large proportion of cases of acute articular rheumatism, so that the infective nature of articular rheumatism may be regarded as all but proved. If it infects joints, why do the marrow and periosteum escape? Still it never suppurates. Several authors have observed cases of osteomyelitis which yielded remarkably to salicylate of soda. There is a suggestion that those which suppurate are called osteomyelitis, and those which do not are regarded as due to acute rheumatism.

The following are points of distinction:—The lesions are usually multiple in acute rheumatism and solitary in osteomyelitis. The pains and temperature yield to salicylate of soda in acute rheumatism, more than in osteomyelitis.

There is marked pain and tenderness over the joint in acute rheumatism, and over the bone in osteomyelitis. Swelling is articular in acute rheumatism and extra-articular in osteomyelitis.

THE PROGNOSIS in acute suppurative osteomyelitis and periostitis varies with the type of the disease, with the initial severity and prominence of the symptoms of intoxication, with the seat of disease—the vertebræ, skull, pelvis, and lower jaw being specially unfavourable—with the multiplicity of the lesions, and the development of internal metastases. One must be on the outlook for the latter, especially endocarditis. The prognosis is favourably influenced by early, accurate and antiseptic treatment.

TREATMENT OF ACUTE SUPPURATIVE OSTEOMYELITIS AND PERIOSTITIS.—This is to be carried out on the same lines as in other suppurative diseases. The strength of the patient is to be kept up with abundant and easily absorbed nourishment. Kocher advocates the liberal exhibition of fluids by the stomach, rectum, or subcutaneous infusion, so as to keep up the blood-pressure, wash out the toxines, and help the functions of the kidneys and skin. He has also found benefit from full doses of salicylate of soda.

Locally, the limb is to be wrapped in a wet antiseptic dressing, and immobilised in some form of splint.

The most important indication is to evacuate the pus as early as possible, and institute drainage to secure its continuous escape, at the same time preventing the entrance of any infection from without, *i.e.* all antiseptic precautions must be observed. So far as the suppuration under the periosteum is concerned, the above indication is fulfilled by a limited incision, it may be with a local anæsthetic, and the insertion of a rubber or glass drainage-tube. Kocher makes the opening with the thermocautery, and endeavours to prevent the access of air to the suppurating area on the ground that oxygen rejuvenates the staphylococci.

The making of a larger incision is indicated if the interior of the bone is to be explored by the trephine, gouge, or chisel.

Many surgeons, especially of the French school, in view of the great predominance of primary suppuration in the marrow, recommend the opening up of the interior of the bone as a routine procedure. Possibly it is better to open marrow needlessly than not to do it when it is necessary. Exploring the marrow does not cause necrosis, and it may prevent it. Others prefer to abstain from opening into the marrow unless there is an indication of its necessity; this may be at the time of incising the periosteal abscess, if there are evidences of suppuration in the interior of the bone, *i.e.* fat droplets in the pus, perforation of the shell of the bone, and pus oozing from the latter, or, in the absence of these indications, on waiting and watching the progress of the disease, the persistence of the fever, and other symptoms of intoxication, indicate that there is likely to be an accumulation of pus in the interior of the bone. One is the less inclined to explore the medullary cavity in deeply seated bones, *e.g.* femur, because the operation is a more formidable one, and attended with considerable loss of blood. Free access cannot be obtained in extensive osteomyelitis of the pelvis, or of the femur, without a severe operation, and the making of a large wound which may be easily infected.

There is more inclination to explore the marrow in superficial bones or segments of bones, *e.g.* tibia, lower end of humerus, parts of fibula, bones of forearm, because in these one is fairly certain of being able to thoroughly clear out purulent areas in the marrow without a complicated wound and much loss of blood. Kocher, Schede, Bergmann, Senn, all agree in confining their immediate medullary explorations to selected cases, and are inclined to the opinion that, employed as a routine method, it is capable of doing as much harm as good. It is also to be remembered that suppuration in the marrow is rarely met with as an actual collection of pus or abscess, more often as purulent infiltration, which has more chances of spontaneous recovery and absorption than an actual abscess. Cases which recover without trephining are fairly often met with; these do not prove that there was no lesion in the marrow, for the latter may be reabsorbed. According to Kocher, when the temperature does not fall after incision and drainage of the periosteal suppuration, one should rather infer the existence of a joint suppuration, or of a metastasis elsewhere, than that the continued pyrexia is due to a lesion in the marrow, because when the periosteal collection is freely opened, the latter tends to be relieved by the escape of pus and toxic products into the external wound through minute canals in the bone.

Where no such secondary suppuration is found to explain the pyrexia, one should trephine the bone and clear out the medulla. The consensus of opinion in this country is decidedly in favour of restricting the operation of opening up the medullary cavity to selected cases, rather than as a routine procedure.

IN THE OPERATION OF TREPHINING THE BONE and clearing out the marrow, the Esmarch's bandage should be omitted because of the risk of embolism and dissemination of infection; a tourniquet is not even required, as there is never any troublesome hæmorrhage. Whether there is a fluctuating area or not, or wherever it is situated, one must remember the frequency with which the disease originates at the juxta-epiphysiary junction, and one should open the marrow there in the first instance with a small trephine. If pus is found, then additional openings may be made according to the extent of the lesion in the marrow, or one may convert the separate trephine holes into one open narrow gutter, with due care not to interfere with the integrity of the shaft. The manipulations inside the marrow must not be rough, for fear of fat embolism; the scraping out of the marrow with a sharp spoon may be overdone. On the whole, when performed in suitable cases, necrosis is less common and less extensive after such operations than without them. It is not to be expected, however, that opening the marrow will always prevent necrosis.

The early or premature *resection of the diseased shaft*, with or without the epiphysis or epiphyses, is very exceptional. It is far better, as a rule, to wait, because one cannot prophesy the extent of the necrosis; the latter is largely a question of the virulence of the cocci. When, however, the suppuration is very abundant, the shaft is extensively bared and appears likely to die, if the bone is accessible and is one of two parallel ones, of which the other will maintain the length and rigidity of the limb segment during reformation, the shaft may be resected with advantage. If the epiphysis is bathed with pus and extensively separated it may be wiser to remove it.

It is of great importance to evacuate all secondary suppurations in the overlying soft parts.

The treatment of the JOINT LESIONS is described in the article on diseases of joints.

Amputation of the limb, which was at one time the routine procedure in suppurative osteomyelitis, is reserved for grave cases in which life is endangered from general infection; where several joints are involved; where there is separation and displacement at the epiphysial junction; where the whole shaft is dying or dead and there are no appearances of a new case, and when the soft parts are extensively involved. That is, apart from the risk to life, when the limb is likely to be useless.

In flat bones, such as the skull or ilium, one must trephine early, because there may be suppuration on both aspects of the bone. In the os calcis, patella, etc., interference must also be early, in order to save the adjacent joints. In the vertebræ, interference is difficult or impossible, and in any case can only be directed to the evacuation and drainage of any abscess which may have formed.

TREATMENT OF THE NECROSIS.—Sequestrotomy, Sequestrectomy. The patient may not come under observation until the acute attack has been recovered from and he applies for treatment of the sequelæ of the disease. So long as dead bone remains *in situ*, so long will there be suppuration from the granulations lining the cavity in which it lies, with discharge of pus externally through the sinuses. The existence of dead bone may be evident to the eye, when it is actually exposed, or it may be inferred by passing a probe down the sinus and striking the bare hard bone.

From external examination it is not always easy to determine the size, character, or number of the sequestra, nor to be certain of their mobility. As regards the best time for surgical interference, the general rule is to wait for spontaneous separation and mobilisation of the sequestrum. In premature operations for necrosis, diseased portions may be left behind; too much bone may be removed, and the subsequent repair may be interfered with. The presence of the sequestrum stimulates and rejuvenates the osteogenetic cells of the marrow and periosteum; the latter, as the result of prolonged irritation, is transformed into a thick tube of cartilaginous consistence already engaged in ossification. While waiting, the drainage may be improved and the septic element combated. Other things being equal, one would wait longer in the case of a limb segment possessed of a single bone, *e.g.* femur, than in a limb segment possessed of two parallel bones. In exceptional cases, where the sequestrum is very slow in separating, *e.g.* outer table of skull, or where the septic risk is a prominent one, or the suppuration so abundant as to affect the health, one may interfere before complete separation and mobilisation, taking care, however, to chisel well beyond the diseased into the surrounding vascular bone.

The operation for the removal of the dead bone (sequestrectomy) consists in opening up the periosteum and the new case to a degree sufficient to allow of the removal of all the dead bone, including the most minute sequestra, as well as any areas of infected granulation tissue and pus. Listerian methods are to be rigidly adhered to. A tourniquet may be employed with advantage. Existing sinuses may be enlarged, but if these are inconveniently situated, *e.g.* in the centre of the popliteal space in necrosis of the femoral trigone, it is better to make a fresh wound down to the bone on that aspect, which affords the best access, and which entails the least injury of the overlying soft parts. The periosteum, which is thick and easily separable, is raised from the new case by suitable elevators, and the new case is freely removed with the chisel or gouge, short of entailing the risk of fracture. In exposing and removing the contained sequestrum, care is taken not to break off any fragment from its worm-eaten edges and extremities, for if any such fragment is allowed to remain, it will interfere with healing, either at the time or at a subsequent period.

In the case of multiple sequestra, several openings will have to be made for their removal. Where sequestra are embedded in an epiphysis, the latter must be freely opened up, or the entire articular end of the bone resected. The dead bone having been removed, steps must be taken to disinfect the cavity in which it lay; to effect this it may be necessary to scrape away the lining granulations with the sharp spoon, and to swab the resulting raw surface with some powerful antiseptic. The cavity may then be stuffed with gauze or worsted, to which iodoform may or may not be added, the stuffing being renewed from time to time until healing is completed. On account of the time and trouble connected with this method of treating bone cavities, other procedures may be preferred. The cavity may be allowed to fill up with blood clot; provided there is perfect asepsis, it may be possible to obtain healing under a single dressing; or skin-bone flaps may be raised from the bone in the vicinity of the cavity and made to cover in the latter, or the cavity may be filled with bone grafts, fragments of living bone obtained in resection of club foot, or fragments of calcined bone, or, lastly, the walls of the rigid cavity may be mobilised, by means of the chisel.

2. SEROUS OR ALBUMINOUS OSTEOMYELITIS AND PERIOSTITIS.—This is apparently the same disease as the preceding, in a mild or benign form; the virulence of the infecting agent (usually the Staphylococcus aureus) being attenuated, the inflammatory exudation is serous or albuminous, and death of the bone is exceptional. There is considerable resemblance to the serous exudation caused by the tubercle bacillus. Like ordinary osteomyelitis, it affects growing subjects between fifteen and twenty years. The periosteal element predominates in the clinical features. The favourite seat is at the ends of the long tubular bones; it is nearly always localised, and there are neither multiple lesions nor metastases.

In the interior of the bone, areas of granulation tissue may be found in the marrow, of an orange yellow colour, along with exudation of serous fluid; when the fluid accumulates in a circumscribed area, the condition has been described as false abscess, lymph abscess, or cyst of bone. The formation of sequestra is exceptional. In the periosteum there may be a similar formation of granulation tissue with exudation of serous fluid between the periosteum and the bone, or between the layers of the periosteum (periosteal ganglion), or in the overlying cellular tissue, in cyst-like cavities. In the adjacent joint there may be exudation of similar fluid (synovitis serosa, hydrops articuli).

Clinically it is characterised by pain, more or less severe at one or other end of the affected bone, and fever, it may be as high as 104° F. at the onset, with rapid defervescence, and the formation of a fluctuating swelling. The inflammatory phenomena are feeble; there is rarely any phlegmon, or reddening of the skin. The fluid rarely comes to the surface and perforates the skin. The effusion may absorb, and the bone may be left more or lsss thickened (hyperostosis). The mildness of the general symptoms in comparison to the considerable exudation is characteristic.

The Diagnosis is only made on puncture of the collection of fluid. It is usually regarded as a hæmatoma or an abscess. If the swelling is incised, there escapes a variable quantity of albuminous liquid, transparent, amber-coloured, viscous, it may be like hydrocele fluid or like synovia; there may be oil droplets from the marrow. The bone beneath is not always bare. There may be a good deal of blood mixed with the exudation, especially in those following more directly on injury.

The prognosis is good, even although the onset of symptoms is quite acute.

Treatment.—If the condition has been diagnosed, it will suffice to evacuate the fluid through a cannula, and inject 10-15 cc. of carbolic lotion (5 per cent), with or without intermediate washing out with saline solution (NaCl ¾ per cent). The injection of the carbolic lotion prevents infection of the track when the cannula is withdrawn. Otherwise the collection of fluid is incised with complete antiseptic precautions, the fluid evacuated, whether outside or inside the periosteum, within the bone or cavity of the joint. If the marrow is opened into, any foci of granulations or minute sequestra should be removed with the spoon. Healing is usually rapid and permanent.

3. GROWTH-FEVER (*Fièvre de Croissance*).—A condition especially described by French authors (Bouilly, Reclus, Poncet), of the nature of an osteomyelitis, unattended with exudation, suppuration, or necrosis, and resulting in the increase of the growth of the bones in length. It may be the explanation of what are popularly known in this country as growing pains, and rheumatism, especially in the lower limbs of children and adolescents.

It is to be regarded as an *attenuated form of staphylococcus osteomyelitis*, because in the same individual one of the affected bones may go on to suppuration or it may be the result merely of a congestion of the marrow occurring during the course of infective diseases. It is not always a true infection of the marrow. Attention is directed to it from the remarkable stimulus which the growth of the skeleton may receive in young subjects during or shortly after an acute illness, *e.g.* typhoid.

Opportunities of investigating the nature of the lesion are exceptional; several authors describe the conversion of yellow into red lymphoid marrow, the greater vascularity of the marrow, especially in the region of the ossifying junction, and in those specially active, viz. those at the knee in the lower limb, and at the shoulder and wrist in the upper limb. It may affect the other bones of the skeleton.

CLINICAL FEATURES.—Two types are recognised, one without fever, and one with (growth-fever proper).

1. *The Non-Febrile Forms.*—The child complains of vague, diffuse, evanescent pains, of lassitude, and of feeling tired in its limbs. The child may be unable to go about. More often it complains of special pains in its limbs, often referred to the joints, after a long walk or violent exercise. The joints are found to be normal; there is tenderness over the epiphysial junctions. Where the epiphysial junction is intrasynovial, as is the case in the hip, the joint itself is the seat of tenderness. There may be pains in the shafts of the bones in relation to exaggerated activity of the deeper layer of the periosteum. The pains may disappear after a night's rest, or last several days, or may come and go over a considerable period. Recovery is the rule with increase in stature. The condition is usually regarded as rheumatism.

2. *The Febrile Form* is more of the nature of an illness, and may be mistaken

for many other febrile conditions. The child is not well, has no appetite, is weary and disinclined to get up. It has pains in its head, and in the vicinity of the joints of its limbs. At times, especially in the evening, the skin is hot and burning; the temperature may rise to 104°. The illness may be sudden and acute, of two or three days' duration, or it may be milder and more prolonged, and there may be relapses, several within a year. The increase in stature usually amounts to four to six inches in a year. The skin of the limbs may present "growth-striæ" (vergetures de croissance), resembling the striæ gravidarum, from stretching of the skin, and tearing of the subcutaneous elastic fibres; they have been observed at the knee, ankle, elbow, and wrist, and are transverse to the long axis of the limb.

In the diagnosis reliance is to be chiefly placed on (1) variable attacks of fever; (2) pains and tenderness at the epiphysial junctions; and (3) increase in stature. It may be mistaken for typhoid and other fevers, tuberculous meningitis, tuberculous diseases of bones and joints. Growth-fever is rarer than any one of these.

Localised forms at the hip and in the spine may be reasonably mistaken for tuberculosis, but this is of less importance, as the treatment is the same for both.

The Prognosis is essentially favourable, but, if carelessly treated, neglected or exposed to injury, it may pass into the more serious forms of osteomyelitis attended with suppuration.

Treatment is directed towards improving the general health by fresh air, sunlight, good food, attention to the bowels, and suitable local treatment during the persistence of the pains. Rest in bed with immobilisation of the affected limb, and later some rigid apparatus, and it may be the application of a blister.

4. Chronic, Persistent, or Prolonged and Relapsing Osteomyelitis following on the Acute, including one Form of Abscess of Bone.—The explanation of the occurrence of chronic, persistent, and of relapsing forms of osteomyelitis is supposed to be as follows:—An acute attack has been recovered from, but has left behind it, in the marrow, foci of infection containing staphylococci which lie latent or dormant, in an attenuated condition, for an indefinite period. At any future time they may be rejuvenated by conditions unknown, aided by depression of the general health, by local injury, strain, or exposure to cold, and cause a fresh outburst of the disease. Although the majority of observers regard these latter outbreaks as remains of the original infection, others believe they may be due to fresh infection, and that the original acute attack is only related to them in so far as it provides a *locus minoris resistentiæ*.

The primary attack may have been a comparatively mild one, and may have attracted little notice, or it may have been one attended with acute illness, suppuration, and necrosis. It may or may not have been efficiently treated. In any case the virus in the marrow has not been eradicated. The nature of the lesion is influenced considerably by the fact that the affected bone has reached maturity, for the disease is one of adult life, and it is further influenced by the changes which the bone sustained as a result of the primary attack, *i.e.* it is not a healthy or normal bone. The lesion may assume the form of *progressive thickening of the bone* (hyperostosis and sclerosis), or that of a *circumscribed collection of pus* (chronic abscess of bone), or that of *ordinary suppurative osteomyelitis with necrosis and sinuses.*

The possibility of the disease running its course without suppuration may be ascribed to the attenuated condition of the staphylococci, and to the greater resistance offered by the tissues of the adult bone to the infecting agent.

While acute osteomyelitis, by its more direct and intense action, arrests ossification, and gives rise to arrests of growth, prolonged and relapsing osteomyelitis, by maintaining less acute action, stimulates growth, and gives rise to thickening and condensation of the bone.

Where progressive thickening of the bone, hyperostosis and sclerosis, is the chief element, the medullary canal becomes obliterated, the bone is increased in girth and weight, the periosteal surface presents the appearances of newly-formed bone, which may assume very varied characters. The periosteum itself is greatly thickened, the muscles of the limb are atrophied, the soft parts may be œdematous, or they may be sclerosed and matted to each other. The skin often presents an unnatural growth of hair. Clinically we may have predominance of the persistent or of the relapsing type of inflammation. There may be one relapse, or repeated attacks over a period of years. They may recur annually at or near the same period. The relapsing type of osteomyelitis is to be suspected in a patient over twenty-five years of age, and the history of an acute attack in youth is to be asked for.

Clinically the local features predominate. This is in marked contrast to the original attack in youth. The pain, swelling, redness, and œdema precede or accompany a moderate degree of fever. The onset of symptoms is often gradual and slow, and the patient may continue his occupation. The patient may call the surgeon's attention to traces of the original attack in the shape of scars, deformity, or loss of function of the limb.

The general symptoms are never so severe, and they rarely make one anxious. The disease is very rarely fatal. The reason of this is partly to be found in the blocking of the medullary canal, the general sclerosis, and the scarring of the soft parts, which all help to circumscribe the infection and prevent the absorption of toxins and any considerable local extension of the disease. Should pus form, the old cicatrised sinuses serve as guides for it to escape externally; an old sinus may actually open up in continuity with the original cloaca in the bone.

The enlargement of the bone occurs especially in the vicinity of the ossifying junction originally diseased; it may appear, however, to be situated higher up the shaft, because of the growth in length of the latter from the epiphysial junction subsequent to the original attack. It may extend along the bone to the next joint. From growth of bone subsequent to original attack, the original scar in the region of the junction may also come to lie higher up the shaft. The thickened bone may be painful on pressure at one special point; pains at night may be the subject of special complaint.

Where there is a circumscribed abscess, it is usually in the interior of the bone at one end of the diaphysis, and the medullary cavity is shut off by new bone. The tibia was affected in 81 out of 113 cases; the lower end of femur and upper end of humerus come next in frequency.

The wall of the abscess may be rarefied; more often it is sclerosed like ivory. It may or may not contain a sequestrum.

Once an abscess is encapsulated it has little tendency to spread or to erupt externally. An abscess supposed to have been in existence for fifty years has been found to contain living staphylococci. Although living, they have lost their virulence in relation to susceptible animals.

The symptoms of abscess are pain which is neuralgic, spontaneous, deep-seated with nocturnal exacerbations. It may be periodic, remittent, and it may radiate throughout the limb. The pain is influenced by congestion of the vessels, because it is influenced by elevation of the limb, and it is relieved by incision of the periosteum, or of trephining the bone, even if no pus is found. Limited tenderness on percussion is characteristic. Swelling or increase in the girth of the bone is a frequent accompaniment. As there may be occasional pyrexia, it is desirable to keep a temperature chart.

Ordinary suppuration with or without necrosis resembles ordinary suppurative osteomyelitis, only it is chronic, or subacute in its onset and progress, and if there be a sequestrum it is buried or walled in and difficult to discover and remove, because it is of old standing and may have lain dormant for years.

These chronic or relapsing forms of osteomyelitis may be attended with *changes in the adjacent joint*, which are liable to be mistaken for tubercle. This is especially true of chronic lesions in the bones in the vicinity of the knee-joint. The author has seen the thigh of an adult amputated for what was supposed to be tuberculous disease of the knee of many years' duration; on section of the lower end of the femur an abscess was found with a contained sequestrum, both dating back to an attack of acute suppurative osteomyelitis which occurred in youth. Such a mistake is to be avoided by more careful search into the previous history of the patient, and valuable information may be obtained from successful X-ray photographs.

The prognosis should be guarded, because one never knows which relapse will be the last, as in the corresponding lesion of the appendix. If rarely dangerous, they are often very persistent in spite of treatment, and may lay the patient up every now and again, and seriously interfere with his occupation. This is especially true of the relapsing disease in the region of the trigone of the femur.

The treatment of chronic, persistent, and of relapsing osteomyelitis varies with the type of the disease. Painless hyperostosis may be left alone.

Where *pain* is the dominant feature one may afford relief by the repeated application of blisters or by incision of the periosteum, or, in more persistent cases, by chiselling into or trephining the bone. Where an *abscess* is suspected in the interior of the bone, the latter must be exposed by suitable incision and elevation of the periosteum; the bone is then explored by means of a drill or trephine. It may be very difficult to discover the abscess, because it may be relatively very small, and because the bone surrounding it is very dense from sclerosis. Having discovered and evacuated the abscess, its cavity is then treated on the same lines as have been suggested for dealing with the cavity in the operation of sequestrectomy.

Should no abscess be discovered, the extensive opening up of the bone required in searching for it will usually result in removing the symptoms.

5. OSTEOMYELITIS WHICH IS CHRONIC FROM THE OUTSET.—The connection of the different members of this group with pyogenic infection has only been recognised within recent years. They were formerly confused with syphilis and tubercle, and without bacteriological examination it is still very difficult to differentiate between them. They may be regarded as including the "ostéite à forme névralgique" described by Gosselin.

In the form of hyperostosis and sclerosis, of ordinary suppurative osteomyelitis with or without necrosis, and of chronic abscess of bone, they correspond in every respect to the chronic forms of osteomyelitis which have just been described as the result of an antecedent acute attack. There is one form, however, which is peculiar to this group, viz. necrosis without suppuration; it therefore requires separate description.

Necrosis without Suppuration—"Quiet Necrosis."—It is a rare disease described by Paget and Morrant Baker, and would appear to be associated with an attenuated form of staphylococcus infection acting over a period of years on the resisting tissues of the bone of an adult. It affects both sexes equally, and may be met with up to the age of fifty or sixty. There may be no discoverable cause of the lesion, or, on going into the history, it may be found to date back to some infective disease, such as typhoid, years before.

Its origin from staphylococcus infection has been demonstrated by Tavel. The disease is probably not so rare as operative experience and museum specimens would indicate, for it is probable that many cases, diagnosed as "chronic inflammation with hypertrophy of bone," may be really of this nature, with a sequestrum embedded in the interior of the bone.

Clinically it is characterised by the development, without any apparent cause, of pain and swelling involving a considerable extent of the femur or tibia of an adult. The pain varies in intensity; it may be continuous or remittent. There is tenderness on pressure. The shaft of the bone is obviously increased in girth, chiefly because it is surrounded by a new case of bone derived from the periosteum. It is not so defined as the swelling in sarcoma, nor does it ever assume the "leg of mutton" shape characteristic of the latter. It resembles sarcoma very closely, however, and, being also liable to spontaneous fracture, the resemblance may be very close. Cases have been recorded affecting the femur of young subjects, in which the bone has presented an abrupt curvature or bend.

The general health is little affected, unless from pain, and pyrexia is only slight and very occasional.

The peculiarities of this form of necrosis, apart from the absence of suppuration, are its extreme chronicity, the incomplete distinction between the dead and the living, the length of time required for the separation of the sequestrum, the absence of any cloaca in the external case of new bone, the formation of new bone both outside and inside in the case of tubular sequestra representing the original shaft.

Many of these peculiarities are illustrated in tuberculous and syphilitic necrosis. They are to be ascribed partly, no doubt, to the fact that the disease is practically confined to the bones of adults, with their peculiarities of structural arrangement and blood-supply, but chiefly to the very attenuated form of the infecting virus giving rise to it, the virus in this respect resembling that of tubercle or syphilis.

The Diagnosis from sarcoma has been referred to in describing the clinical features. Further points in favour of the inflammatory lesions are the relief of pain by resting in bed with the limb elevated, the absence of dilated veins underneath the skin, and the more uniform, hard, incompressible nature of the swelling. The X-rays may reveal the presence of the sequestrum.

The diagnosis from syphilis will rest largely on the history of the patient and on the effect of treatment.

The diagnosis from tuberculous disease of the bone is so difficult that, as a rule, it will only be made on exploring the bone and on examining the diseased tissues microscopically or bacteriologically.

The Treatment, in most of the recorded cases, has consisted in amputation of the limb. It may be possible, however, to attempt the removal of the sequestrum on the usual lines. The symptoms will be relieved in any case by trephining the bone.

II. Osteomyelitis and Periostitis due to Pyogenic Infections other than Staphylococci

It has been assumed that the majority, including all the typical forms of osteomyelitis and periostitis, are the result of infection with the Staphylococcus aureus. Infection with other organisms is exceptional, and the resulting diseases resemble very closely those due to staphylococci. *Every micro-organism with pyogenic properties may cause osteomyelitis.*

Staphylococcus albus.—For the experimental production of osteomyelitis with the white staphylococcus, a larger dose is required than of the yellow variety, and the pus is whiter. Clinically the general opinion appears to be that the disease is milder and less likely to be followed by necrosis. *The citreus* appears to be intermediate between the aureus and the albus.

Streptococcus pyogenes is rarely met with as a pure infection. It has been especially met with during infancy, and in the newly born, where the mother suffered from erysipelas or other infection. The onset of the illness may be very acute, but if there be no general septicæmia, the high fever subsides in a few days, when the pus forms. Joint lesions are more common than those in the bones. In the latter, as compared with staphylococcus osteomyelitis, the pains are less severe; suppuration is earlier, more diffuse and abundant; the skin may be red, as in erysipelas, and the glands may be enlarged. Necrosis and separation of epiphyses are less frequent and less extensive. The pus is not so thick nor is it of a greenish colour.

PNEUMOCOCCUS.—Although medullary infection with pneumococci was first identified in association with pneumonia, it has been met with apart from pneumonia and from meningitis, especially in the earlier years of childhood. Infection is supposed to enter through the lung. It is relatively benign, for although the general symptoms may be grave to start with, the local lesions are less severe, as if the virulence of the infection were soon exhausted. Joint lesions are fully more common than those in the bones. The latter have been met with in relation to simple fractures. Incision of the purulent collection in relation to the bone is usually sufficient, as there is little tendency to the formation of sequestra. Some slight thickening of the bone, or a few adhesions in the adjacent joint may remain, otherwise recovery is complete.

THE TYPHOID BACILLUS.—Suppuration in connection with the bones during or after typhoid fever have only been differentiated since bacteriological investigation has shown which lesions are due to the typhoid bacillus, and which are due to infection by other organisms, *e.g.* the Bacillus coli, the Staphylococcus aureus. The occurrence of mixed infections, and of infections by other organisms than the typhoid bacillus, is sufficiently accounted for by the ready means of entrance afforded during typhoid fever,—erosions in the mouth, nose, and throat, blisters and sloughs of the skin, and ulcers in the intestine. During typhoid fever, the bone-marrow is markedly congested, and appears to favour the lodgment of the bacillus in its tissues; thus lodged, it is capable of persisting for long periods until some injury or other factor determines its return to active life. It has been found in pure culture in the marrow seven years after an attack of typhoid fever.

The lesions in the bones may or may not be attended by suppuration, and they may involve the marrow, or the periosteum, or both. They may be solitary or multiple. Their favourite seat is in the ribs, at the costo-chondral junctions, and in the tibia. Where unattended by suppuration, the lesion may recover completely, or apparently recover, and then recur. When pus is formed, it presents, on examination, a pure culture of the typhoid bacillus. The abscess may be dumb-bell shape, one part under the periosteum, and the other in the marrow; the two communicate with each other by a hole or sinus in the shell of the bone. Some authors describe the action of the typhoid bacillus on the tissues as necrosing rather than pyogenic, resembling in this respect the tubercle bacillus; the exudation is then largely composed of granulation tissue which undergoes pseudo-caseation, and conversion later into a brown or red syrupy liquid.

CLINICAL FEATURES.—The bone lesions are much more often a sequel of typhoid than of occurrence during its course. The majority occur during the seventh or eighth week. They are never met with in individuals who have not had typhoid fever. They are more often met with under the age of thirty.

The symptoms are almost entirely local. There is little disturbance in the general health; fever may be absent altogether. The symptoms develop insidiously in the shape of vague pains in the bones. After being in several bones, they usually become localised in one part of the skeleton. They are aggravated by movement, or by handling the bone, and they are worse at night. They may disappear spontaneously, but reappear again later.

There may be redness and œdema of the overlying soft parts. Swelling may form with vague fluctuation over the bone.

On incision there escapes a yellow creamy pus, or a brown syrupy fluid.

The lesion appears to be purely periosteal in the majority of cases. Complete resolution is common enough. Some authors have observed resolution of the suppuration, after drawing off some of the pus through a trocar and cannula.

Simple incision of the periosteum is more often practised, and results in recovery. The joint may fill with pus, and yet recover completely. Some thickening of bone may persist, or there may form a superficial sequestrum. Death of the bone is exceptional.

DIAGNOSIS.—Where the abscess develops very slowly like a cold abscess, or where no pus is formed, the condition may resemble tuberculous disease of the bone. Stress is to be laid on the history of typhoid, or the recognition of its presence, and on the bacteriological examination of the pus or other fluid obtained on puncture or on incision of the periosteum.

PROGNOSIS.—In pure infections with the typhoid bacillus it is favourable. It may be regarded as fortunate that the bone complication usually occurs after subsidence of the fever. Recovery is assured, but is apt to be slow; a relapse is not uncommon.

The prognosis is less favourable, if the infection of the typhoid bacillus is complicated with other organisms, *e.g.* the Streptococcus pyogenes.

TREATMENT.—Aspiration of the pus or incision of the periosteum is usually sufficient. Some recommend the clearing out of the wall of the abscess in which the bacilli are lodged. In the cases of the ribs, it may be necessary to resect portions of them. In the rare cases in which the suppuration is in the interior of the bone, it may be necessary to open into the marrow.

COMBINATIONS OF THE PRECEDING ORGANISMS.—*Mixed Infections; e.g.* Staphylococcus aureus and albus, Staphylococcus aureus and colon bacillus, Staphylococcus aureus and streptococcus. Typhoid and colon bacilli or typhoid with staphylococci or streptococci. Very little is known of them, but they are usually regarded as graver.

Infections following the Eruptive Fevers (scarlet, measles, smallpox, etc.) are usually secondary infections by staphylococci, streptococci, and other organisms, gaining entrance through lesions in the mouth, throat, or skin.

III. PYOGENIC DISEASES OF BONE FROM DIRECT INFECTION

In all the pyogenic diseases considered in the preceding sections, infection has been assumed to have reached the bones through the arteries. There still remain for discussion those which are the result of direct infection, through a wound involving the bone, *e.g.* compound fractures, gunshot injuries, osteotomy, amputations, resections, operations for ununited fracture, in all of which the marrow may be exposed to any infection of the wound in the soft parts. In the case of the jaws, the infection may spread to the bone from lesions of the mouth. In the case of the skull, the infection may spread to the bone from lesions of the scalp, or from lesions of the bones themselves, *e.g.* syphilis, tumour.

The lesions included in this group are much rarer since the introduction of Listerian surgery. Museums are full of specimens dating from former times.

The direct infections differ fundamentally from the indirect ones. The patient may be otherwise healthy. The organisms are those infecting the wound. The lesion is exclusively or mainly an osteomyelitis, which commences and is always most intense at the site first infected, *i.e.* in the marrow exposed in the wound. It may spread for some distance along the medullary canal.

The marrow presents changes of ordinary septic or suppurative inflammation. There is rarely an accumulation or collection of pus within the bone. There may be fungating granulation tissue protruding from the exposed marrow. If necrosis takes place, it is usually limited.

In amputation osteomyelitis the sequestrum may be annular, affecting the cross section of the bone at the saw line, or may be tubular, extending up the shaft, tapering off above, the upper end of the tube being represented only by the thin shell of bone next the medullary canal.

The periosteum chiefly participates in forming new bone, in being more easily detached, and in being thicker than normal. In the macerated specimen the new bone may present a characteristic coral-like appearance, and may be perforated by cloacæ.

The relations of the disease to pyæmia are well known as resulting from the septic phlebitis in the marrow.

In compound fractures the disease may lead to death of the fragments and the formation of sequestra, which are included or walled in by new bone, hence persistent sinuses may result, with thickening of the adjacent periosteal surface. (See Compound Fracture).

The clinical features of osteomyelitis associated with wounds are those of ordinary septic infection, rigors, etc., with sweating and depression. In addition to suppuration in the original wound there may be secondary abscesses along the shaft of the bone, and these may communicate with the central sequestrum through cloacal apertures in the new case.

TUBERCULOUS DISEASES OF BONE

These are the result of infection of the marrow and periosteum by tubercle bacilli which have been conveyed to these tissues through the arteries.

In contrast to the more typical pyogenic diseases they are characterised by their *painless and insidious onset,* and by their slow progress and chronicity. They are further characterised by their frequent localisation in the less active red marrow of the short and flat bones, and in the epiphyses of the long bones rather than in their ossifying junctions. Their localisation in the epiphyses explains the frequency with which the infection spreads to the adjacent joint, and the reason why tuberculosis is of much greater importance as a disease of joints than a disease of bones. In contrast to the pyogenic and syphilitic infections, tuberculosis is comparatively rare in the shafts of the long bones with the exception of the phalanges, metacarpals, and metatarsals of children.

Although the site of entrance and method of infection by tubercle bacilli are rarely apparent on clinical observation, pathological experience tends to show that tuberculous disease of bone is exceedingly *rare as a primary affection,* if indeed it occurs at all, and that the bacilli are nearly always derived from some pre-existing focus in the body, namely the lung, the bronchial, mesenteric, retro-peritoneal, cervical glands, or in the genito-urinary organs. The importance of this fact lies in the necessity of our regarding a patient with tuberculous disease of a bone, however localised, as the bearer also of an internal source or store of tubercle which may then, *or at any future time,* lead to an outbreak in some other part of the skeleton or elsewhere in the body; and it is also to be borne in mind that the original source or store of tubercle in the lung, gland, or elsewhere may itself develop fresh activity and menace the life of the patient. In this connection it is interesting to note that recent work by Stockman would appear to indicate that the bone disease formerly considered to be due to phosphorus is really of tuberculous origin.

As was assumed in pyogenic diseases of bone, so also in tubercle we must believe in the *capacity of the bacilli of lying dormant* in the marrow for indefinite periods. We must also recognise the influence in the production and localisation of tuberculous disease in bone of such additional factors as *special tissue predisposition* and *the influence of injury.* The injury which is related to tuberculous disease is usually insignificant in character; it is to be taken into account not only in the determination and localisation of the disease to start with, but as a factor in its aggravation, and as a factor also in the breaking out afresh or relapse of old and apparently cured lesions, it may be after the lapse of many years. The influence of age is well recognised as a predisposing factor in tuberculous disease, at the same time the apparent overwhelming frequency during the early years of life is to be largely accounted for by the correspondingly larger number of individuals alive during these earlier years. The fact must be emphasised that *tuberculous disease may be met with at any period of life*, because the statement is so often heard from the uninitiated that a given lesion cannot be tuberculous because the patient is advanced in years.

Age also influences the localisation in certain parts of the skeleton; without going into details, it may be said that the hip, spine, and phalanges attain their maximum in the earlier years of life, while disease in the knee, elbow, shoulder, and wrist is comparatively frequent in adults. The influence of sex appears to be largely one of liability to injury. Before the age of ten and after sixty, during which periods of life the male and female are equally exposed, there is less predominance of the male sex than between the ages of ten and sixty, during which the male is more exposed to injury (Cheyne).

The influence of heredity, preceding debilitating disease (measles, whooping-cough, pneumonia), climate, faulty hygiene, overcrowding, and poverty, is in bone similar to their influence in other tissues, and need not be further referred to in this place. Only a *résumé* of the individual lesions will be given, as they will be considered in detail under the different regions (*q.v.*)

I.—TUBERCULOUS DISEASE IN THE PERIOSTEUM—TUBERCULOUS PERIOSTITIS.
    Bones specially affected.
    In superficial bones, e.g. *rib*, *ulna*.
    Clinical features, diagnosis and treatment.
    In deeply seated bones, e.g. *femur*, *vertebræ*.

II.—TUBERCULOUS DISEASE IN THE INTERIOR OF BONES—TUBERCULOUS OSTEOMYELITIS.
    General characters and clinical relationships.
    Tuberculous dactylitis.
    Osteomyelitis in other bones, e.g. *femur*, *tarsus*.

I. PERIOSTEAL TUBERCULOSIS—TUBERCULOUS PERIOSTITIS.—This is less common as a primary disease than as an affection secondary to lesions in the interior of bones.

It is typically met with in the ribs, vertebræ, cranium, bones of the face sternum, and in the long bones of the limbs.

Clinically the development of tuberculous granulation tissue in th deeper layer of the periosteum, and its subsequent caseation and lique faction is attended by the insidious development of a doughy swellin which is usually painless although distinctly tender on pressure. It i often discovered by the patient accidentally and its date of origin mis-state As the swelling increases in size it tends to become boggy, to fluctuate, an to assume the characters of a cold abscess; the latter usually perforates th fibrous layer of the periosteum and invades and infects the overlying so parts; its spread in the soft parts is influenced by gravity, and by the an tomical arrangement of the tissues. The size of the abscess is no indicati of the extent of the lesion from which it originates. Finally the co abscess reaches the deep surface of the skin; the latter becomes dusky r or livid, it becomes thinned and finally gives way. The contents of t cold abscess then escape through an aperture in the skin which is called t sinus. If a probe is passed down the sinus it strikes the friable worm-eaten bone, which, from its undergoing disintegration, is commonly described as being in a condition of caries. The sinus tends to persist so long as any active tubercle remains in the tissues from which the cold abscess took its origin. If left to itself it readily allows of the entrance of infection from without, and this favours the persistence and extension of the tubercle both in the bone and in the overlying soft parts. The orifice of the sinus is occupied by pouting, flabby granulations; the edges of the orifice and the skin may be undermined or they may be actually infected with tubercle which may spread to the skin in the immediate vicinity like lupus. There is little new formation of bone on the periosteal surface in the vicinity of the lesion compared with that observed in staphylococcus lesions and in syphilis. A cold abscess may form between the ribs and the pleura.

The features of tuberculous periostitis in other superficial bones, *e.g.* tibia, ulna, sternum, skull, are very similar to those observed in the ribs. Now and then one may find small superficial sequestra of the surface compact bone embedded and surrounded by the tuberculous granulation tissue.

Diagnosis of Periosteal Tubercle in Superficial Bones.—Before the formation of the cold abscess, the localised thickening which develops over the bone is to be differentiated from *syphilis*, from *chronic forms of staphylococcus osteomyelitis*, and from such rare conditions as *lipoma* or *ganglion*, and from *periosteal sarcoma*. From the last mentioned the diagnosis may be especially difficult; apart from differences in the history, in the predisposition, and in the constitution of the patient, sarcoma is characterised by its more rapid and continuous progress, by the severity and persistence of pain in spite of rest, by the more tumour-like character of the swelling, by the absence of suppuration, and of any benefit from treatment. A rise of temperature in the evening is more likely to be present in cases of rapidly-growing sarcoma than in tubercle, unless the latter is complicated with pyogenic infection. In difficult cases recourse may be had to the X-rays or to exploratory incision, and, if necessary, histological and bacteriological examination of the diseased tissue.

When met with in the stage at which the cold abscess has burst and left a sinus, the diagnosis is to be made from staphylococcus osteo-myelitis which has been recovered from and which has left a sinus; the history of the illness, the seat of the lesion, the appearances of the external opening of the sinus, and the conditions found on probing the latter, are to be carefully taken into account. The diagnosis, however, may only be cleared up by an examination of the diseased tissues after their removal.

The Treatment of Tuberculous Periostitis.—Apart from that of tubercle in general, it is to be conducted on the usual principles. In the superficial and therefore accessible bones to which, so far, we have been confining our attention, it will usually assume the form of direct surgical interference with the diseased tissues. Where what is called an operation is objected to, and especially where the disease has reached the stage of cold abscess, the liquid contents may be drawn off through a cannula and an emulsion of iodoform injected in its place. On the whole it is more satisfactory to expose the infected tissues by a free incision at the earliest possible stage of the disease, and especially before the skin is infected or perforated; the diseased tissues are then thoroughly removed with a spoon or gouge, and the area of the wound thoroughly and mechanically cleansed with gauze; the bleeding having been arrested, the exposed tissues are swabbed with pure carbolic acid, and it may be also with iodoform. If the skin be perfectly healthy the wound may then be stitched up and primary and satisfactory union may be anticipated. If, on the other hand, the skin be infected, and especially if there be superadded pyogenic infection from without through a sinus, it is best not to close the wound but to stuff it with iodoform worsted which is to be renewed at intervals of several days until sound healing takes place from the bottom.

In deeply seated bones, such as the *femur*, tuberculous disease in the periosteum may not reveal itself at all until the formation of a cold abscess in the overlying soft parts, and the true nature and situation of the disease may only be recognised on incising the latter and exploring its interior with the finger.

In the vertebræ, in which periosteal tubercle is fairly common, the disease may spread along the surface of several bodies, lifting up the periosteum and anterior common ligament and invading the surface bone. It reveals itself clinically by the formation of abscess when this has attained sufficient size to be capable of recognition, and it may be also by the development of curvature of the spine, which, however, is never so pronounced nor so angular as when the bodies themselves are the original seat of the disease.

In periosteal tubercle of any bone, but especially *in the region of the articular ends of the long bones*, there is considerable risk of the direct extension of the disease to the adjacent joint. This is very common. The symptoms of tuberculous disease of the joint may be the first indication of the periosteal lesion, hence the necessity of the earliest possible recognition of the latter, and of its radical treatment before infection of the joint has taken place.

II. Tuberculous Lesions in the Interior of Bones.—*Tuberculous Osteomyelitis.*—It is difficult to give a systematic description of these which will be of any clinical value to the practitioner, because in the great majority of cases they are only recognised when some joint or periosteal lesion to which they have given origin is explored in the course of an operation, or when the bones in which the lesion is situated have been removed (in an excision or amputation), and laid open for purposes of examination. It is only in the case of bones which are quite superficial, *e.g.* phalanges, ulna, clavicle, skull, lower jaw, that tubercular disease in the marrow gives rise to definite physical signs, which will allow of its clinical recognition. In other situations, *e.g.* in the vertebra, the existence of tubercular osteomyelitis can only be inferred from the development of a pronounced curvature of the spine, or of an abscess which from its situation and associations is probably of spinal origin. In relation to the joints, the diagnosis of tubercular disease in the interior of the adjacent ends of bones can only be inferred from the persistence of the joint disease in spite of treatment, from its graver character and from the earlier formation of a tuberculous abscess. It is quite safe to say that there are many individuals going about with a tuberculous lesion in the bone marrow, the existence of which is entirely unsuspected, for it may remain latent for an indefinite period, and may even undergo cicatrisation and spontaneous cure without ever having given rise to any symptoms whatever. The general statement may also be made that, with few exceptions, tuberculous disease in the interior of a bone does not give rise to any symptoms until by extension it reaches one o other of the surfaces of the bone affected. If it reach the periosteal surface of a superficial bone, it will lend itself to recognition *as a periosteal swelling* which may afterwards develop into a cold abscess. In a deeply-situated bone such as the femur, an *abscess in the overlying soft parts* may be th first indication of the disease, and until this abscess is opened and explore its origin from a lesion in the interior of the bone may remain unsuspected and unknown. If the lesion in the marrow erupts on the periosteal surface where the latter is in immediate relation to the reflection of the synovial membrane of the adjacent joint, it *may infect the synovial membrane* and spread along the latter. Lastly it may erupt on an articular surface and directly *infect the cavity of the joint as a whole* (*vide* "Joints").

A description of the different anatomical forms of tubercular disease in the marrow is beyond the scope of the present article. It may be briefly stated, however, that while certain of them are characterised by the same caseating tendencies which are so familiar in tuberculous disease in other tissues, others are peculiar to the interior of bones, inasmuch as they may be attended with sclerosis of the bone within the limited area affected, and that such sclerosed bone may perish under the toxic influence of the tubercular virus and lead to the formation of a characteristic sequestrum.

DIFFUSE FORMS OF TUBERCULOUS DISEASE IN THE MARROW (TUBERCULOUS OSTEOMYELITIS PROPER) are rare, unless we include under this head the extensive tuberculous infiltration and caseation of the marrow observed in the interior of phalanges, metacarpals and metatarsals. This is the condition known as *tuberculous dactylitis.* In this the marrow is replaced by tuberculous granulation tissue, the shell of the bone is excavated from within, while new bone is formed from without, very much as in the growth of central sarcoma, and the bone may present the appearance of having been expanded. This is the spina ventosa of the ancients. It may be limited to one end of the bone, or may extend throughout its entire length. At any stage in the disease, death of the shell of the bone may take place with the formation of a sequestrum which may consist of the greater part of the shaft, more or less altered by changes which preceded its death; that is to say, it may be sclerosed in some parts, rarefied or perforated in others, and may present very little resemblance to the normal bone from which it was derived.

Clinically tuberculous dactylitis is much more easily recognised than lesions in the marrow of other bones; the bone being superficial there is visible increase in its girth, the swelling is usually spindle-shaped; at any time there may be perforation of the shell of the bone, the tubercle then invades the periosteum and overlying soft parts, and gives rise to a cold abscess and subsequent sinuses.

There may be difficulty in diagnosing between the dactylitis of tubercle and that of syphilis, and especially in recognising tuberculous dactylitis in a child presenting evidences of inherited syphilis (*vide* "Syphilis").

The Treatment of Tuberculous Dactylitis is influenced by the knowledge that spontaneous recovery is by no means uncommon, and that the functional results may be very much better than those following upon operative interference, even although the finger may be permanently dwarfed. Aim at improving the general health and temporise with some placebo. The finger may be immobilised in plaster of Paris, or a blister may be applied. Operative interference is indicated if a cold abscess forms, or for the removal of a sequestrum where there is a persistent sinus. Amputation may be advised in the case of toes; in the fingers it is rarely called for unless the disease is threatening to spread up the limb, along the lymphatics, or is otherwise endangering the general health. It will be more often required in adults than in children.

In the larger long bones of the limbs diffuse lesions in the marrow are comparatively rare. The clinical features and anatomical changes associated with them correspond in the main to those described in the phalanges, except that, inasmuch as the bones are more deeply situated, there is greater difficulty in their recognition and in their differentiation from other chronic enlargements of bone, *e.g.* pyogenic osteomyelitis, tumour, syphilis, hydatids. The nature of the disease may only be recognised on exploring the enlargement of the bone by operation or by successful X-ray photographs. The treatment will consist either in clearing out the disease in the interior of the bone or in amputation.

In the short bones such as those of the *tarsus*, diffuse infiltrating tubercule is also met with; clinically it assumes the features of joint disease or of cold abscess, and the exact nature of the lesion is only recognised on exposure and section of the affected bone or bones. It is usually an indication for amputation, as several bones and joints are simultaneously affected; if the disease is less extensive, removal of the bones and tissues actually infected may suffice.

SYPHILITIC DISEASES OF BONE

These are the result of the infection of the marrow and periosteum by the syphilitic virus which has been conveyed to these tissues through the arteries. They are met with at any period of life subsequent to the original contagion. The graver forms of bone disease are chiefly met with in the advanced stage of acquired and inherited syphilis. Although it must be assumed that the virus is carried to all parts of the skeleton, the actual development of the disease is powerfully influenced by local predisposition on the part of individual bones, and by such external factors as exposure to injury.

In point of frequency, syphilitic diseases of bone are much more rarely met with in practice than those due to pyogenic or tuberculous infection. They are also characterised by their predilection for certain bones which are rarely involved by the latter, by the predominance of disease of the shaft of the bone over disease at the articular ends, and by the rarity of joint complications.

THE EARLY LESIONS OF ACQUIRED SYPHILIS.—*Evanescent periostitis.*—These are met with during the period of the early skin eruptions. The patient complains of pains over the frontal bone, ribs, sternum, tibiæ, and ulnæ. The pains are distinctly worse at night while the patient is in bed. There is localised tenderness, elicited by tapping the bones mentioned. The patient may be unable to bear the pressure of a hat on the forehead. Swelling of the periosteum may be recognised on palpation; it rarely amounts to what may be described as a periosteal node. It is possible that the more deeply seated pains in the bones occasionally met with in early syphilis may be due to similar changes in the marrow. These early superficial lesions never suppurate; they usually disappear without leaving any trace of their existence; they may, however, leave localised periosteal thickenings which may long remain painful and tender on pressure; it is possible that such thickening on the inner aspect of the skull may be the cause of persistent headache. The diffuse encrustation on the surface of bones preserved in our museums may result from this condition. Apart from their tendency to spontaneous recovery, they further resemble the accompanying lesions in the skin and lymphatic glands, in the rapidity with which they yield to specific treatment; where they resist mercury alone, relief is obtained by the addition of potassium iodide.

THE LATER LESIONS OF ACQUIRED SYPHILIS—*Gummatous periostitis and osteomyelitis.*

Pathological Anatomy.—Like the corresponding lesions of the soft parts, gummatous disease of bone is characterised anatomically by the formation of embryonic or gummatous granulation tissue in the periosteum and marrow in the form of circumscribed masses or as a diffuse infiltration. This tissue leads to structural alterations in the bone in relation to it, of the nature of rarefaction or disappearance of the osseous tissue in the first instance and of condensation and new formation of bone at a later period, or these two sets of changes may go on simultaneously side by side. The persistence of the disease, it may be over many years, for it has little tendency to spontaneous recovery, may lead to profound alterations in the structure and architectural arrangement of the bones affected. Actual destruction and death of the bone is associated with degenerative changes in the gummatous tissue, more especially where it caseates, liquefies, breaks down, and erupts externally on the skin surface, and there is superadded pyogenic infection. The destruction of bone may be in the form of molecular disintegration of the osseous tissue, so that it escapes externally in small particles mixed up with the discharge (syphilitic caries) or in the form of visible portions of bone (syphilitic necrosis). These destructive changes may affect certain parts of a bone, while the remaining portions may be simultaneously the seat of the merely structural alterations already referred to.

The bones specially affected are the skull, septum of nose, nasal bones, palate, tibia, femur, radius, ulna.

In the skull the actual separation of portions of bone as sequestra is exceptional; it may affect the outer table alone, or the entire thickness of the bone or an entire cranial bone. It may be due to the gummatous infiltration itself, encircling the bone, and isolating it from its blood supply; it is helped by extreme sclerosis of the adjoining bone strangulating its capillary network, and still more by the entrance of pyogenic or septic infection from the overlying broken skin. The latter would appear to be the chief factor in the actual death and separation of bone which has been encircled by gummatous granulation tissue, for, when the skin is unbroken such isolated bone may remain indefinitely, at any rate for years, embedded in the granulation tissue as an aseptic foreign body. If incompletely encircled by granulation tissue, even where there is superadded pyogenic infection and suppuration, its separation is extremely slow, partly from the want of vascularity in the sclerosed bone round about, and partly from the density of the bone to be separated.

In the septum of the nose, nasal bones, and palate, the gummatous granulation tissue infiltrates the periosteum diffusely, eats into the bone with which it is in contact, softens and ulcerates on the mucous surface, and becomes complicated with pyogenic infection. The result is a crumbling away of the affected bones in particles or visible portions (sequestra) and the production of deformity.

In the long bones the tibia is the one specially affected, and may be taken as the type.

Circumscribed masses of gummatous granulation tissue in the deeper layer of the periosteum may organise and lead to the formation of a *well-defined node*, or a series of these may lead to an *irregular nodular thickening* of the bone, which at the same time will be rendered denser and heavier from proliferative changes in the bone adjacent to the gummata. Or the gumma may undergo degeneration and liquefaction, and may rupture on the skin surface and lead to similar changes to those described in the skull.

A circumscribed gumma in the marrow may similarly organise and lead to *formation of new bone* which obliterates the medullary canal, or it may degenerate and erode the bone (like the spina ventosa of tubercle) to such a degree that it *undergoes spontaneous fracture*, or the shell of the bone may be perforated on one of its aspects, the gummatous tissue comes to the surface, erupts through the periosteum and finally through the skin, so that again there may be the same changes as those described in the skull.

There may be *multiple gummata* scattered up and down the marrow which may individually pass through one or other, or combinations of the changes which may attend the solitary gumma.

If the gumma involve the articular end of the bone *it may involve the adjacent joint* and give rise to syphilitic arthritis.

The changes described as met with in the tibia or any long bone may persist, or may come and go over a period of years, with alternating stages of absorption, and destruction, and of new formation of bone, both outside and inside the shaft, so that finally *the appearances and architectural arrangement of the bone may be profoundly altered.* . . . *If the bone be still growing*, as is so commonly the case in inherited syphilis, the alterations in the contour, shape, and structure of the bone will be still more pronounced, and will persist throughout life as permanent evidences of the disease. The sabre-blade deformity of the tibia (of French authors) has this complicated origin; the bone appears to be curved with the convexity forwards, the result of bossing or heaping up of bone along the anterior edge; at the same time it may be thickened, sclerosed, and it may be also lengthened. The loss of the crest of the shin may be more evident in other cases, the anterior surface of the bone being flat or rounded.

CLINICAL FEATURES OF THE LESIONS IN ADVANCED SYPHILIS.—The pains are much more intense than during the so-called secondary period, and are described as boring as if a gimlet were being driven into the bone. They are worse at night, and may prevent sleep. They have been ascribed to the compression of the nerves of the bone as they pass through the sclerosed haversian canals, and marrow generally. The bones on inspection may present evidences of the various anatomical changes described above; the bones involved being superficial, they lend themselves to visual and digital examination. The occurrence of a circumscribed smooth swelling is characteristic of the *periosteal gumma*. The centre is soft and elastic; the margins are firm, hard, and shade off into the surrounding bone. The skin is free over the swelling. Recovery may ensue with or without the formation of a densely hard node, or the gumma softens in its centre, the skin becomes adherent, thin, and red, and finally gives way, and a thin watery fluid escapes. Through the opening one may see the tough, chamois-leather-like gummatous tissue. The opening in the skin persists as a sinus, or it becomes an ulcer with irregular crescentic margins; a probe reveals the presence of disintegrating bone (caries) or an actual sequestrum (necrosis). There is superadded pyogenic infection.

The disease is most inveterate; changes in the direction of repair and of separation of a sequestrum are extremely slow.

Persistence of discharge impairs the general health and may result in waxy degeneration in the viscera. The injurious influence on the health is chiefly the result of superadded pyogenic infection and the absorption of toxines. The waxy disease is recovered from, if the bone disease is cured.

Central gummata in the long bones may not reveal their presence unless they give rise to spontaneous fracture, or until they invade an adjacent joint or until they erupt through the shell of the bone and reach the periosteal surface. In the latter case there may be the same sequence of events as described in the periosteal gumma.

The Deformities of the long bones have been sufficiently described.

In the skull, in addition to the changes which are common to it and the long bones, there are certain special features. Where there is considerable new formation on the internal surface of the skull there may be pressure on the brain and consequent headache, epilepsy, paralysis, or pressure on nerves, especially at their point of exit from the skull. Where gummatous disease has burst on the skin surface and there is superadded pyogenic infection, there may be not only the most persistent sinuses or ulcers with sequestra, but there may be serious intra-cranial complications of the nature of abscess, meningitis, or pyæmia.

In the nose, the disease may be characterised by gradually increasing deformity, by stinking discharge, by epiphora, lachrymal abscess, and fistula; *in the palate* by the formation of perforations and consequent alteration of speech; *in the wall of the orbit* by exophthalmos and paralysis of the muscles of the eye.

The diagnosis of the syphilitic nature of any given disease of bone is materially assisted if the practitioner entertains a suspicion to this effect, for his attention is then directed to the fact that the disease has selected certain situations, to the history of the patient, and to search for other evidences of the disease.

The diseases most liable to be mistaken for it are *chronic staphylococcus osteomyelitis, sarcoma, and tuberculosis.* In the latter, assistance will be derived from remembering its frequent localisation in the epiphyses, the absence of nocturnal pains, the frequent termination in suppuration, and the inefficacy of specific medicines. Syphilitic diseases of bone are much less common, they more often involve the shaft of the bone, are more often periosteal, and they rarely involve adjacent joints.

TREATMENT.—*In the stage of gummatous granulation tissue* the internal administration of mercury and of iodide of potassium, along with the local application of blisters, is usually followed by the rapid relief from pain, and the disappearance or absorption of the gummatous tissue. A periosteal gumma need not be incised, even although it is softened and the skin is red over it.

In advanced cases, where the bone has undergone structural alterations (hyperostosis and sclerosis), or where there are sinuses, suppuration, and necrosis, the specific remedies may have little effect either in alleviating the symptoms or in bringing about recovery, or may only afford relief during their administration, and the disease relapses as soon as they are left off. The general health must be improved. A sea-voyage or a sojourn at some resort such as Aix-la-Chapelle may be recommended.

In cases of persistent or relapsing gummatous disease of the bone with ulceration of the skin over it, along with the constitutional treatment, it is better to adopt the same methods of surgical treatment as have been recommended in tuberculosis. The diseased soft parts are to be removed with the spoon and scissors, and the diseased bone gouged or chiselled away, including any sequestrum and some of the sclerosed bone round about.

Where there is merely *hyperostosis and sclerosis* of the bone, with severe pain which does not yield to blisters, the thickened periosteum may be incised and the sclerosed bone perforated with a drill, gouge, or trephine.

In rapidly breaking down gummatous disease in the nose or palate, which is often so persistent, and which results in serious deformity, surgical removal of the affected tissues may check the progress of the disease and lead to recovery. In the case of the nose, the author has carried this out by splitting the nose in the middle line, removing the infected parts, and scrubbing the raw tissues with pure carbolic acid; the wound is then dusted with iodoform and stuffed with gauze.

LESIONS OF THE BONES IN INHERITED SYPHILIS.—The bones are more often affected in the inherited than in the acquired disease. As a result of the infection being inherited there are certain lesions of the skeleton, especially during the earliest months of infancy, which do not occur in the acquired disease. Further, those which are common to the acquired and inherited types present certain peculiarities both in their progress and in their results, because the bones affected are engaged in growth, and have not attained maturity. The individual lesions will only be briefly referred to in this place, as they will be described in the general article on inherited syphilis.

During the earliest months of life the infant may present—

1. *A porous friable encrustation or irregular layer of osteophytic new growth on the periosteal surfaces of the shafts of the long bones;* this condition is only recognised clinically in the superficial bones of an emaciated infant. Whether it is not partly or entirely the result of coexisting rickets is open to question; the same doubt has been thrown on the two following lesions.

2. *A similar encrustation of new bone on the outer surface of the flat bones of the skull*, especially along the sutures. If this take place along either side of the sagittal suture, the latter appears as a furrow or cleft between the bosses on the parietal bones; the so-called nateform skull. Or the new formation of the bone may be arranged symmetrically around the anterior fontanelle, giving rise to an appearance which has been compared to that of a hot-cross bun. The new bone is at first very vascular and spongy, but may become dense and hard. They are recognised clinically as firm rounded bosses or elevations, and are known as Parrot's nodes. The centres of ossification of the bones usually escape. More rarely there may be a diffuse sclerosis of the entire skull, with or without premature union of the sutures and arrested growth of the skull.

3. A condition known as *Craniotabes*, in which the osseous tissue is absorbed in limited and usually multiple areas and the cranial bones revert to their primary membranous condition. It is chiefly observed in the occipital region, and is recognised clinically by the dimpling of the skull in the areas involved under the pressure of the finger as if it consisted of thin cardboard. Its occasional independence of rickets is maintained; where due to syphilis alone, it recovers under mercurial treatment.

4. A condition at the ends of the long bones known as *syphilitic epiphysitis* or osteo-chondritis syphilitica. It is usually met with in infants from one to three months old. It essentially consists in the formation of a soft semi-fluid granulation tissue of a yellowish colour, which replaces the spongy bone at the ossifying junction, and causes a certain degree of swelling or bulging at the junction of the shaft with the epiphysis. This swelling is smooth and globular; it may be chiefly on one aspect or surround the junction like a collar or ring. The affected limb hangs inert, and is incapable of voluntary movement. Hence it has been called syphilitic pseudo-paralysis. The reaction of the muscles is not impaired. The foot assumes the attitude of equino-varus, the hand is pronated and flexed. It may affect one ossifying junction or several simultaneously. It may result in partial displacement and mobility at the ossifying junction; this is not attended with actual crepitus but an obscure grating may be elicited. Curious malpositions of the limbs may result from epiphysitis with partial displacement, which may be accompanied by irregular or stunted growth from interference with the ossification. The adjacent joint may be filled with fluid.

Suppuration only occurs if there is superadded pyogenic infection.

Recovery takes place if early treatment is instituted; this includes that of syphilis and immobilisation of the limb.

The lesion is diagnosed from the swellings at the ossifying junctions in rickets, by its occurring before the age of six months, by the swelling being less uniform and less symmetrical, by the absence of the rickety rosary, and by the improvement which follows the exhibition of mercury.

5. Other and rarer lesions. The *ilium*, *scapula*, and *vertebræ* may present similar changes to those described in the long bones, both in relation to their epiphysial junctions and on their periosteal surfaces. A condition of *fragilitas ossium* has been described, in which there may be one or more greenstick fractures from slight violence, with little or no attempt at union. *In the ribs*, at the junction of the anterior and middle thirds, the bone may be replaced by a gelatinous marrow of a transparent yellow colour; this may result in their spontaneous fracture.

The treatment of the preceding lesions is that of inherited syphilis (*q.v.*)

The Lesions of the Bones in the Later Stages of Inherited Syphilis.—These, which may occur at any time from the commencement of the second dentition up to maturity, correspond to the later or tertiary lesions in acquired syphilis. They differ from the latter inasmuch as they involve bones engaged in active growth, they are met with fully more often at the ossifying junctions than in the shafts of bones, they may interfere with the future growth of the bones affected, and they are not nearly so amenable to the influence of iodide of potassium as those occurring in the tertiary manifestations of adults. As in the acquired disease, they may persist, or may come and go over periods of many years. Their anatomical and clinical features, as met with in the skull and in the long bones of the limbs, have been described in acquired syphilis.

SYPHILITIC DACTYLITIS is a characteristic clinical type and merits special description. Although met with in acquired syphilis it is more common in the inherited form, and in the latter may be met with in infancy or at any time up to twenty years.

It affects the fingers more often than the toes, and both of these more often than the metacarpals or metatarsals. It is commonest in the region of the first phalanx of the index or thumb. It may affect several fingers and toes at the same time or in succession, one after another. Anatomically the lesion consists in a gummatous infiltration of the soft parts surrounding the phalanx or in a gummatous osteomyelitis and periostitis, or a combination of these.

The segment of finger affected becomes very gradually swollen; the swelling is more evident on the dorsum than on the palm, and it terminates abruptly at the metacarpo-phalangeal joint. According to the distribution and extent of the gummatous disease the swelling may be acorn-shaped, fusiform, or cylindrical. The swelling is firm and elastic, and but slightly painful or tender. The movements of the finger are impaired especially if the disease involve one or more of the joints. In its early stages it is very amenable to treatment, and may recover and leave a perfectly normal finger.

If left untreated, the degenerated gummatous tissue may approach the surface. The skin becomes thin, purple, and gives way, often on the lateral aspect of the finger, and a sinus persists. The disintegrated bone may crumble away and escape in the discharge; the formation of a visible sequestrum is exceptional. Loss of bone may result in shortening and deformity of the finger.

The condition, especially in its early stage, is to be diagnosed from chondroma, and from tuberculous dactylitis; the latter is often difficult, and may be attained only by watching the effects of treatment. Skiagraphy may be instructive and helpful in recognising tubercular cases where the phalanx is hollowed out and "expanded" in the form known as spina ventosa; in syphilis it may show more sclerosis, and more new periosteal bone.

The diagnosis and treatment of the lesions of the bones in the later stages of inherited syphilis is to be approached on similar lines to the acquired disease. Reference has already been made to the comparative inefficacy of the iodide of potassium. Inveterate cases of bone disease from inherited syphilis are to be dealt with on very much the same lines as cases of tuberculous disease: improvement of the general health by country or seaside air, by good food, by the administration of cod-liver oil, iron, and arsenic, and local surgical measures, consisting in the removal of gummatous granulation tissue and diseased bone where there is no prospect of spontaneous recovery.

TUMOURS OF BONE

Primary tumours of bone grow from the marrow, periosteum, and, in the case of young bones, from cartilage; they are all of the connective tissue group, and include the types known as fibroma, myxoma, lipoma, chondroma, osteoma, and sarcoma. *Secondary tumours* include all those which are capable of giving rise to metastases in bones (carcinoma, tumours of thyroid), and those which spread to bones from other tissues by direct contiguity (epithelioma).

They are distinguished, according to their seat, into periosteal and medullary.

Periosteal tumours are usually lateral in the first instance, but they may completely surround the bone; they are bounded externally by the fibrous layer of the periosteum: the latter may, however, be invaded and perforated by the cellular elements of a malignant tumour; in the long tubular bones the thick periosteum may materially retard the eruption of the growth into the overlying soft parts, whereas the thin periosteum of the flat bones, *e.g.* skull, has but little influence in this direction.

Central or medullary tumours, whether circumscribed or diffuse, usually lead to cellular absorption of the bone in immediate relation to the tumour, while new bone is laid down externally. The bone as a whole may be very greatly increased in girth as a result of these processes, and is often described as having been "expanded" by the growth in its interior. Whether a central tumour erupts or not on the external surface of the bone, depends on the rapidity of the growth of the tumour and on the activity of the periosteum in forming new bone. The latter is greater in the long bones of the limbs than in the flat bones of the skull or trunk.

Primary tumours of bone are usually solitary, but the chondroma, osteoma, and sarcoma may be multiple from the outset.

Regarding the etiology of tumours of bone it is worthy of remark that they may follow upon injuries, and that they are specially liable to develop in situations, *i.e.* in the region of the ossifying junction, in which there may have occurred some irregularity in the process of ossification, and in which residues of the tissues concerned in the process may have remained unused and in an embryonic and unstable condition.

PRIMARY TUMOURS: OSTEOMA.—The true osteoma or bony tumour is to be differentiated from the new formations of bone which may result from irritation or inflammation, and from abnormal ossifications, *e.g.* those occurring in muscles, tendons, etc.

The osteoma is very much more commonly periosteal than central. It is called an *exostosis* when it projects from the surface of a bone. Those derived directly from the tissues of the periosteum may be described as *membranous exostoses*, *e.g.* those of the skull; they are often dense or compact like ivory, and are covered only by the fibrous layer of the periosteum. When derived from hyaline cartilage they are known as *cartilaginous exostoses*, *e.g.* those at the ends of the long bones of the limbs; the latter resemble normal bone in structure, and are covered or invested with a layer or cap of cartilage from which they may continue to grow until the growth of the skeleton is completed. Because of their spongy or medullated structure they may be described as *cancellous exostoses*. The hyaline cartilage from which they develop may result from a metaplasia of the tissues of the periosteum; more commonly it is derived from remains of the epiphysial cartilage or of the original cartilage of the embryo.

Exostoses are usually rounded tumours of limited size, sometimes pedunculated or mushroom-shaped; their free surface is smooth or nodulated. Cartilaginous exostoses in the vicinity of joints may be invested with a synovial sac or bursa, (*exostosis bursata*), derived either from the synovial membrane of the adjacent joint with which its cavity may communicate, or of adventitious origin. It has been observed to contain cartilaginous loose bodies.

Clinically the osteoma is met with as a hard indolent tumour, growing from a bone and fixed to it. It is usually discovered accidentally, unless it presses injuriously on adjacent structures. Hence its special features vary with its situation. In the orbit it will displace the eyeball, in the nasal fossæ and in the external auditory meatus it causes obstruction which may be attended with ulceration and discharge, in the vicinity of a joint it may interfere with the movements of the latter, on the inner side of the knee it may prevent the patient grasping the saddle in riding; when growing from the dorsal aspect of the terminal phalanx of the great toe, it displaces the nail and may project through its matrix at the top of the toe, while the soft parts over it may be ulcerated from pressure; when growing in the vicinity of the nerves of a limb it may press on these and cause pains, cramps, and other symptoms.

The diagnosis of osteoma will be arrived at from their long duration and slow growth, by their situation, and by their hardness. The X-rays may be of assistance.

Treatment.—An osteoma which does not cause symptoms may be left alone. The cartilaginous varieties will cease growing at maturity; if causing symptoms they are removed by dividing the neck or base of the tumour with a strong chisel. The dense varieties especially met with in the bones of the skull present greater difficulties. The base or neck of the tumour is to be perforated in many directions with highly-tempered drills, rotated by some form of mechanical engine.

The multiple exostoses of the skeleton met with in childhood and youth are ossified chondromata. They are of congenital origin, and are often inherited. They correspond to the normal apophyses of bones and have been likened to the branches of a tree. They may interfere with the growth of the skeleton; they themselves cease growing when the latter is completed. They are chiefly met with at the ends of the long bones which possess the most active epiphyses, *i.e.* in the region of the knee and at the shoulder and wrist. They may also grow from the flat bones of the trunk, *e.g.* scapula, pelvis. They are often symmetrical. They vary in size from a hazel-nut to a fœtal head. When they originate from the ossifying junction of a long bone they may migrate along the shaft as the latter increases in length; when they originate from the region of the epiphysis itself they remain at the end of the bone. Their clinical features and treatment are the same as the solitary cartilaginous exostosis.

CHONDROMA.—The chondroma, which undergoes ossification, has been described under osteoma. Other chondromata are rare.

It may grow from the periosteum (perichondroma) of the scapula, ilium, ribs, long bones of the limbs, forming tumours which early project from the surface of the bone, and may attain an enormous size. Or they may grow in the interior of a bone, the so-called enchondroma; they may lie latent for a considerable period, replacing the old bone, while new bone is formed outside by the periosteum; further growth of the tumour results in bulging of the bone attended with increase in its girth; they may finally erupt through the external shell of the bone and project from its surface, with displacement of the overlying soft parts.

The cartilage composing the tumour frequently undergoes degeneration. Myxomatous changes result in softening of the cartilage into a glairy semi-fluid jelly; if this change takes place throughout the tumour, the latter may present the appearances of a cyst. On the other hand the cartilage may become harder from calcification and from conversion into bone, which may resemble that normally formed from cartilage more or less closely. The most important transition of all is that of the chondroma into sarcoma, the so-called *malignant chondroma* or chondro-sarcoma; it is associated with the reversion of the cartilage to an embryonic type and the acquisition of greater vegetative activity, so that it may recur on removal, and parts of it may be carried off in the blood stream and give rise to secondary growths, especially in the lungs.

Clinically the typical chondroma is met with as a visible, slowly-growing tumour. It is especially met with in the bones of the hand, often in a multiple form. Its surface may be smooth or lobulated. Its consistence may be dense and elastic like normal cartilage, or it may present areas of softening or of bone-like hardness. The soft parts move freely over it, except in relation to the superficial bones of the fingers, where the skin may become adherent and ulcerate and suggest a malignancy which does not exist. In more deeply seated bones, the chondroma is not recognised so easily and may be confused with sarcoma. When of large size they may press upon nerves or upon vessels, and may interfere with the movements of joints.

Their origin may be ascribed to injury. According to Virchow they are more often met with in those who have suffered from rickets during childhood.

The Prognosis varies with the type of tumour: so long as they are innocent they are very innocent, and grow very slowly or may even become stationary. Anything like active growth is an indication of malignancy, and they are then very malignant.

In the Treatment of chondromata it is necessary to insist on the removal of the whole tissue of the tumour, even although it appears to be innocent. In periosteal growths, especially if they are pedunculated, this is comparatively easy, unless an entire bone such as the scapula is involved, in which case it may be better to remove the bone as a whole. In the case of central tumours the external shell of bone is to be removed over an area sufficient to allow of the enucleation of the tumour in the interior. It may be better practice to resect the affected portion of bone, or even to amputate the limb segment, especially in the case of the fingers. Should there be any suspicion of malignancy, early and high amputation is the procedure recommended.

FIBROMA is chiefly met with as a periosteal growth from the bones in relation to the mouth and pharynx, of which the simple epulis of the gum and the naso-pharyngeal polypus are well-known examples. They may be very vascular and cellular, and may show all transitions to sarcoma, or they may be very fibrous, and they may undergo partial conversion into bone. Their diagnosis and treatment belong to the special regions in which they are met with.

MYXOMA, LIPOMA, AND ANGIOMA are all very rare. They may be periosteal or central. Their exact nature is seldom recognised unless they are subjected to operative interference.

SARCOMA is the commonest tumour of bone, and is therefore of great clinical importance. Two main types are to be differentiated according to their origin in the interior of the bone or from the periosteum.

Central or medullary sarcomata originate in the marrow, and replace the spongy bone in their vicinity without giving rise to any changes in the exterior of the bone for a variable period. At a later stage the bone becomes enlarged or "expanded" and the tumour tissue may be merely invested by a thin shell of new periosteal bone, or may erupt into the surrounding tissues. The tumour tissue usually respects the articular cartilage of the adjacent joint.

The type of tumour-tissue varies: it may be a soft, rapidly-growing, small round-celled sarcoma, or a firmer, more slowly growing spindle-celled sarcoma (fibrosarcoma), or it may present a marked tendency to undergo ossification (ossifying sarcoma), or its vessels may undergo special development, so that it may pulsate, or it may largely consist of large giant cells; the latter, which has long been regarded as a sarcoma and been named myeloid, is now placed in a class by itself, especially by French writers, because of its innocent characters.

Where the cells are arranged in alveolar spaces in the stroma, the condition may merit the designation of alveolar sarcoma, but may be more accurately classified with the endotheliomata.

The softer and more rapidly growing sarcomata are liable to undergo degenerative changes and pigmentation from hæmorrhage. Liquefaction of the tumour tissue may occur with the formation of cyst-like spaces.

Periosteal or peripheral sarcomata originate in the periosteum, usually on one aspect of the bone in the first instance; later they tend to surround the bone and give rise to a spindle-shaped thickening or tumour. The tumour tissue is most often spindle-celled, but may be round-celled or mixed. They vary considerably in their progress: some are characterised by their tendency to erode the bone underneath, others by the amount of new bone which they may form; this new bone may amount to an actual frame-work or skeleton for the growth; it usually assumes the shape of spicules radiating from the surface of the bone from which the tumour has originated. Where the formation of new bone is a prominent feature, the tumour may be described as an ossifying sarcoma. Chondro-sarcoma has been referred to under chondroma. Sooner or later the tumour tissue tends to erupt through the fibrous layer of the periosteum, and infects the overlying soft parts, infiltrating or displacing these, until finally it reaches the skin; when the latter gives way, the tumour tissue protrudes as a fungating bleeding mass.

Clinical Features of Sarcoma of Bone.—They are met with more often before the age of thirty, and are comparatively common in children. Males are more liable than females in the proportion of two to one, partly because of their being more exposed to injury.

Their most common seat is in the long bones of the limbs, and especially at the juxta-epiphysial junctions in the region of the knee-joint in young subjects. The upper end of the humerus and upper end of the fibula come next in order of frequency. Apart from the bones of the limbs, they are met with in the jaws, in the skull, in the ribs, scapula, pelvis, etc.

The objective symptoms consist in the appearance of a tumour connected with a bone, which increases progressively in size. It may be on one aspect or may surround the bone; at the end of a long bone it acquires the shape of a leg of mutton. Its surface may be uniform or bossed; its consistence varies with its situation, whether central or periosteal, with its structure, and with the changes which it has undergone. The co-existence of solid and of fluctuating areas is characteristic. On firm pressure, fine crepitation may be felt from crushing of the delicate framework of new bone, or there may be parchment-like or egg-shell crackling from yielding of the thin unresisting shell of bone under the pressure of the fingers. There may be arterial pulsation, and a bruit may be heard. The patient often complains of pain which is aggravated by exertion, and which is worse at night. The pain is said to be more severe in central tumours before they erupt on the surface of the bone.

The soft parts over the tumour may for a long time preserve their normal characters; they may become œdematous, and the dilated subcutaneous venous network becomes very evident through the thinned, transparent skin.

The *adjacent joint* usually remains intact, but its movements may be impaired from the mere bulk of the tumour, or from effusion, or from portions of the tumour tissue pushing themselves into the cavity of the joint. Inflammatory changes may occasionally occur, leading to stiffness and adhesions, and it may be vicious attitudes of the limb, *e.g.* flexion at the knee. The local elevation of temperature over the tumour, which may amount to 2° or more, is a point of diagnostic importance.

When the overlying soft parts are invaded, they become matted to each other and adherent to the skin; the latter is thinned, distended, and finally sloughs. The tumour tissue then protrudes, fungates, ulcerates, breaks down, bleeds, and decomposes. The amount of bleeding may be serious. These changes are sometimes hastened by careless puncture of the swelling for purposes of exploration. *Spontaneous fracture* may be an early manifestation of the disease, especially if it involve the shaft of the femur or humerus; it follows slight violence, and it is unattended with much ecchymosis or swelling; it never really consolidates, although there may be an attempt at union by the formation of a cartilaginous callus. The deformity and other signs resemble those of traumatic fracture.

Suppuration may occasionally take place in the soft parts over the tumour, although the skin remains unbroken. This may be incapable of explanation unless an exploratory puncture has been made without precautions. The lymphatic glands in the vicinity may be enlarged without being actually infected with sarcoma, for they have been observed to recover after removal of the primary growth. Actual infection of the glands may occur in spite of the sarcomatous nature of the lesion. As the tumour progresses the patient emaciates and fails in health and appetite. There is evening pyrexia, apart from ordinary septic complications. The patient is worn out with suffering, want of sleep, discharge, loss of blood, etc., and finally succumbs, with or without evidences of secondary growths in distant parts. These are especially met with in the lung or pleura, but may occur in other organs or in other bones, or may be generalised throughout the body.

The Diagnosis of Sarcoma of Bone is often difficult in the early stage in which it may be of real value. The following are the conditions with which it is liable to be confused:—

1. *Chronic Staphylococcus Osteomyelitis at the end of a long bone:* in sarcoma the swelling is more irregular, it is better defined above and below, because the adjacent bone is healthy, whereas in the inflammatory lesion there is hyperostosis gradually tapering off along the shaft; in tumour also, the adjacent joint is less often implicated, and there is greater liability to spontaneous fracture.

2. *Disease of the adjacent joint,* especially while swelling of the knee, may closely simulate sarcoma, and the two conditions have been mistaken for each other. It is difficult to state rules which may be of use in practice, but attention is to be directed to the history of the patient and of his disease, to the shape and exact seat of the swelling, to the absence of suppuration, and to the fact that the joint disease will yield to rest and other methods of treatment which will not influence the progress of a sarcoma.

3. *Innocent tumours* (*chondroma*) *and cysts* (*hydatid*).

4. *Tuberculous or syphilitic swelling of the bone.*

5. *Secondary cancer* may present precisely the same appearances as a primary sarcoma of bone, and may evade diagnosis altogether unless its connection with a primary growth elsewhere (liver, breast, thyroid, prostate, rectum) is recognised.

In all of the above, assistance may be obtained from the X-rays, o recourse may be had to exploratory incision with microscopical examinatio of the suspected tissue.

The Prognosis of sarcoma of bone varies—1. *With their malignancy,* o which the readiest clinical indication is the rapidity of their growth befor removal. 2. *With their situation;* there is little hope where the skul spine, or pelvis is involved, because their removal may be out of the questio and because of the damage to the adjacent viscera; in the limbs the progn sis is graver the nearer the disease is to the trunk. 3. *With their situatio relative to the bone;* other things being equal, the periosteal are mo dangerous to life because so many of the central tumours are myeloid i character, and therefore comparatively or actually innocent. It is n possible, however, always to differentiate clinically between periosteal ar central tumours. The latter are more often at the ends of bones, are bett defined, are spherical or triangular in shape, and may yield egg-sh crackling on pressure. The former more often involve the shafts of bones, are ill-defined, and are spindle-shaped or fusiform. 4. *The stage at which they are subjected to operative treatment.*

The Treatment of Sarcoma of Bone in the case of those which lend themselves to operative interference is removal at the earliest possible moment. Until recent years the invariable practice has been to perform amputation well above the growth where one of the bones of a limb is involved. This is still to be recommended as a routine procedure, with the single exception of myeloid tumours. It is only fair to add, however, that conservative operations have been performed, and in some instances with success, even in the case of cellular periosteal sarcomata (Mikulicz). Each case is to be dealt with on its own merits, and according to the operative experience of the surgeon.

If amputation is contemplated, the supposed sarcoma should always be explored just before its performance to make certain of its necessity. Another knife must be employed for the amputation to prevent the risk of implanting the cells of the tumour.

As regards the amputation itself, it is not essential to go as high as the joint above, provided the tissues are divided well beyond the tumour; skin-flaps with circular division of muscles is the method of choice. In cases where removal of the tumour can hold out no prospect of permanent recovery (*e.g.* where there are secondary growths), relief from suffering, discharge, or hæmorrhage may be a sufficient indication for amputation, and in cases where pain is a prominent feature it may be worth while to remove the tumour, or even to trephine the shell of the bone.

The operative treatment of sarcoma of the bones of the head and trunk is considered in the sections dealing with these special regions.

The myeloid tumour of bone—often called myeloid sarcoma—is characterised by the enormous giant cells which form the most conspicuous element of the tumour issue. They are met with in the same situations as sarcoma. They grow very slowly, or, after attaining a certain size, may become latent. The general health remains intact; secondary growths are quite the exception. In other respects the clinical features closely resemble those of a slowly growing central sarcoma of a long bone already described.

Their prognosis is much more favourable than sarcoma.

Their treatment is to be conducted on conservative lines; it resembles that of tubercle in bone rather than sarcoma. The tumour must be extirpated, either by a process of digging out from the interior of the bone or by resecting the portion of bone affected. Amputation is only indicated if the whole end of a bone is destroyed, which is essential for the integrity and usefulness of the limb.

Pulsating or Erectile Tumours of Bone—Aneurysm of Bone.—The infrequency with which this condition is met with is responsible for the obscurity which exists concerning its nature. It would appear to be the result of special changes in an already existing tumour, sarcoma, endothelioma or myeloma. The tumour tissue may largely or entirely disappear, while the vessels and vascular spaces of the tumour undergo a remarkable development. The tumour may come to be represented by one large blood-containing space communicating with the arteries of the limb; the walls of the space consist of the remains of the original tumour, plus a shell of bone of varying thickness. The favourite seat of the condition is the lower end of the femur, the upper end of the tibia, and the bones of the pelvis.

The clinical features are those of a pulsating tumour of slow development; the patient may or may not have observed the original stage in which the tumour was solid; there may be considerable complaint of pain. The tumour may attain such dimensions that it may no longer suggest to the observer that it is of bony origin, unless he is able to recognise the parchment or egg-shell crackling on forcibly pressing on it with the fingers. As in true aneurysms, the pulsation (and bruit) disappear on compression of the main artery above, but in the spurious aneurysm of bone, the distension of the tumour and the pulsation are longer in returning when the pressure is removed. In the region of the knee it is easier to differentiate between the two, because one is able to make out the relations of the tumour to the bones and to the popliteal artery; in the groin and pelvis the diagnosis may be extremely difficult. Aneurysms of bone, especially those with a shrill bruit, have been known to consolidate and disappear.

The treatment is influenced by the malignant or innocent nature of the original tumour. In malignant cases involving the bones of the limbs, amputation is indicated; in the pelvis operative treatment may be out of the question. In innocent tumours one may consider the possibility of a conservative operation, resembling that known as the "old operation" for true aneurysm.

SECONDARY TUMOURS OF BONE.—These include all varieties of tumours which are capable of causing metastasis in bone, and those which spread to the bone by direct contiguity.

I. *Metastatic Tumours.—Secondary cancer* is a comparatively common disease in bone. Its characters depend on the nature of the primary growth which gave rise to it. There are softer forms which grow rapidly, and which are characterised rather by the eating away of the bone. In more slowly growing forms there may be considerable formation of a new imperfectly organised bone, often very deficient in lime salts, so that the affected bones may be cut with a knife; this condition may be widely diffused throughout the skeleton and has been described as cancerous osteomalacia. Secondary cancer of bone may not reveal itself clinically at all, or it may suddenly attract notice from the occurrence of spontaneous fracture; this specially occurs in the shaft of the femur or humerus and in the vertebræ; in the latter situation it is attended with a painful form of paraplegia which may involve the lower or all four extremities. On the other hand, the disease may show itself clinically as a tumour of bone, it may be of considerable size, which may be mistaken for a primary sarcoma, unless the existence of the original cancer is evident or suspected, and is then discovered on examination.

The cancers specially liable to give rise to metastasis in bone are those of the breast, liver, uterus, prostate and intestine.

Secondary tumours derived from the thyroid gland require special mention, not only because of their frequency, but because they are peculiar inasmuch as neither the primary growth in the thyroid nor the secondary growths in the bones are necessarily malignant. They are therefore amenable to surgical treatment. *Secondary sarcoma,* whether derived from primary growths in the bones or in the soft parts, are very much rarer than the corresponding cancers. They are not to be confused with cases in which sarcoma is generalised throughout the skeleton from the outset. They are of little clinical importance, as with isolated exceptions they are quite out of the range of operative treatment.

II. *Cancer of bone resulting from direct extension from adjacent soft parts.*—In this group, the bone is involved by the spread of the cancer in the same way as any other tissue. There are two clinical types.

The first is met with in relation to *epitheliomata of mucous surfaces, e.g.*

palate, gums, antrum, frontal sinus, auditory meatus and middle ear, and will be described under these special regions, and in relation to epithelioma and rodent cancer of the skin over any bone.

The second is met with in relation to *epithelioma when it attacks a persistent sinus* resulting from suppurative osteomyelitis. The sinus may have existed twenty or more years. The disease originates at the external or skin orifice of the sinus, and spreads down to the bone and into its interior. Inside the bone the progress of the cancer is resisted by the sclerosed condition of the bone, and the obliteration of the medullary canal; still, although its progress is slow, the infiltration of the bone is often more extensive than would appear externally. It is recognised clinically by the characteristic epitheliomatous growth in the sinus and its vicinity, and by the stinking discharge from the bone. The operative treatment is influenced by the extent of disease in the soft parts overlying the bone, and will consist accordingly in wide removal of the diseased tissues or in amputation.

CYSTS OF BONE.—With the exception of the parasitic cysts, which are considered elsewhere, cysts in the interior of the bone are usually the result of the liquefaction of tumour or other solid tissues; the tumours which may result in the formation of cysts are the chondroma, the myxoma, and the sarcoma. Cysts are also formed in relation to osteomalacia, and to the rarer forms of attenuated staphylococcus infection.

Special epithelial cysts derived from the teeth are met with in the jaws, and are described under this head.

MULTIPLE MYELOMA.—Under this vague heading, brief reference may be made to a rare form of disease of bone which is accompanied by albumosuria. Originally described by Bence Jones, cases have been recorded recently by Bradshaw, Senator, and others. The lesion in the bones has been variously described as primary multiple sarcoma, myeloma, or lymphoma, and as partaking of the characters of osteomalacia. It would appear to consist in the development, in the interior of the ribs, sternum, and vertebræ, of multiple foci of a vascular cellular tissue or new growth, leading to absorption of the framework of the bone, and rendering it liable to spontaneous fracture. The external appearances of the bones are not altered; there is an absence of anything capable of being recognised as a tumour. This condition will be further referred to in the consideration of albumosuria under the disorders of the urine.

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